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THE DETERMINATION OF SERUM AND URINE SALICYL LEVELS DURING TREATMENT WITH SALICYL DRUGS

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The initial purpose of this study was an attempt to improve the method of Coburn-Brodie[1, 2] for the determination of salicyl radicals in the blood. In an article about the treatment of rheumatic fever[4] with high detoxified doses of salicylates, I pointed out that it is of great importance to check the blood and urine salicyl levels during treatment. Without such a control it happens too often that the prescribed doses are not taken by the patients. Repeatedly I heard that the treatment with high doses of salicylates was declared ineffective, whereas a simple check of the blood and urine salicyl levels would have discovered fraud or negligence or bad absorption of salicyl compounds, given in tablets. These causes of low salicyl levels in the blood can easily be distinguished from temporary hypersecretion of the drug by the kidney which lowers, of course, also the serum salicyl levels.

My experience with the Coburn-Brodie method was that the results were often lower than was expected. This fact was also observed by the late pediatrician Chester Stewart. In an attempt to discover the cause of these observations, I tried several modifications of the Coburn-Brodie method. I replaced the ethylen-dichloride extraction which is part of that method, by protein-precipitation, using the blood flitrate. During these investigations which were done shortly after the publication of Coburn's article in 1943, that is four years ago, I made an interesting observation which led to the development of a new, much simpler method which is now regularly used in laboratories of Charity Hospital, New Orleans. Accidentally a drop of a solution of ferric nitrate fell into the clear serum of a patient who was treated with large doses of salicylates. The serum showed a strong brown-violet color. I decided to examine the possibility of replacing the extraction procedure and the deproteinization procedure by a method, based upon this accidental finding. I examined first a large number (about sixty) of sera of patients who had had no drugs during 48 hours. Never a trace of a color-reaction was seen with the ferric nitrate solution. On the other hand, if the patient had taken small amounts of salicyl compounds, one hour before the drawing of the blood, the color-reaction appeared with ferric nitrate, but not yet with the extraction-method. Therefore the here described method is more sensitive.

PRINCIPLE OF THE HERE DESCRIBED METHOD

To 0.5 c.c. of clear non-hemolytic serum is added an iron-nitric acid reagent. If the serum contains more than 4 mg. per cent salicyl radicals, a color appears which is brownish in the lower and violet in the higher concentrations. In spite of these two colors, the optical density in a photoelectric colorimeter, produced by increasing salicyl concentrations, increases in a straight line. The cause of this unexpected fact is appar-

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ently that all the colors are mixtures of brown and violet.

Accurate determinations are made with a photoelectric colorimeter, (abbreviated PEC). A standard curve, prepared with standard solutions of sodium salicylate, has to be made for every PEC, because with three PEC of the same make, I found differences up to 20 divisions with the same colored serum. The preparation of the standard solutions and the calculation will be described in a more extensive article which will be published in one of the next numbers of the Acta Medica Scandinavica. Approximative estimations can be made with the aid of a color-chart which will soon be available in stores of laboratory supplies.

REAGENT

Only one reagent is necessary. After many trials the following mixture was chosen. Weigh out five grams of ferric nitrate. Dissolve this in 98 c.c. of aqua dest. Add 2 c.c. of concentrated nitric acid. (The sequence of additions is of importance). This reagent will be called: "Iron Reagent" or I. R.

PREPARATION FOR THE UNKNOWN SERUM

For making the mixture the following sequence is necessary:

Iron reagent	0.3 c.c
Aquae dest.	7.2 c.c.
Serum	0.5 c.c.
(clear, non-hemolytic)	

No filtration is necessary, if clear serum is used. If the serum is cloudy by some mistake (*vide infra*), then it is better to repeat the drawing of the blood, but approximative results can be obtained by filtration of the mixture through filters which remove very small particles, for example, Whatman filter No. 5 or S & S 602. Hemolytic serum should never be used. In the beginning of this investigation, I received several samples of cloudy or hemolysed serum. I could eliminate these mistakes by sterilizing the syringe and needle in hot air or in an autoclave but not by boiling, and by heating the tubes previously in order to remove the moisture on the inside.

Serum gives more reliable results than plasma, because I saw that different amounts of anticoagulants, present in the plasma, may influence the color formation. Allow five minutes for the development of the color and read within the next few hours. The blood should be *always* drawn *just one hour after the latest dose of the drug*, in order to be able to compare the results with determinations on other days. At this time the "serum-salicyl" is about at its highest point.

In using the color chart, mentioned above, the tube is placed at an angle of 45° in a special rack which allows the light to fall just behind the colored fluid. (This special rack with a color-chart will be available as laboratory supply). If a PEC is present in the laboratory and a standard salicyl curve is made, it takes, of course, only little more time to obtain very accurate results.

In the more extensive article (*vide supra*) a table of observations will be published, showing a comparison between the results here described method and the Coburn-Brodie method. The results of the Coburn-Brodie method were 7 to 18 per cent lower than those with the here described method. Such large differences in the results explain the inconsistency which was also observed by the late Dr. Stewart, as mentioned above. Another table in that article shows that the results with the here described method are about the same in cases treated with or without the addition of sodium bicarbonate. This contradicts the publication of Smull et al.[3] which was the impulse of a nation-wide epidemic of intravenous injections of salicylates, without bicarbonate, in order to reach higher blood salicyl levels. Several patients died from this treatment. Also the observations of Huntington et al.[5] show that the results of Smull et al. are erroneous.

DETERMINATION OF THE URINE-SALICYL LEVEL

In this article only the principle of this determination can be mentioned. For details I refer to the extensive article, mentioned above. The principle is just the same as it is for serum salicyl determinations. The 0.5 c.c. serum is only replaced by 0.05 c.c.

of urine and as result is reported the quantity "urine-salicyl" excreted in the two-hour period, following the administration of the drug. This is necessary in order to eliminate the influence of the diuresis. Routine examinations of "urine-salicyl" are not yet described in the literature, as far as I could ascertain. This simple determination may replace a large part of the serum salicyl determinations, in a similar manner as is the case in diabetes mellitus with the glucose of urine and blood.

REFERENCES

1. Coburn A.: Salicylate therapy, Johns Hopkins Hosp. Bull., 73:435, 1943.
2. Brodie B. et al.: The determination of salicylic acid in plasma, J. Pharm. & Exp. Ther., 80:114, 194.
3. Smull, K. et al.: Effect of sodium becarbonate on salicyl level, J. A. M. A., 125:1173, 1943.
4. Peters, Johan T.: The necessity and possibility of giving detoxified large oral doses of salicylates in the treatment of rheumatic fever (To be published soon in the Acta Medica Scandinavica).
5. Huntington, R. et al.: Studies in rheumatic fever, Ann. Int. Med., 24:1029, 1946.

STERNAL MARROW IN HEALTH AND DISEASE

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The sternum as a source of bone marrow possesses many advantages:

Accessibility: The sternum is a wide, flat, elastic bone and is practically subcutaneous. In the subcutaneous tissue there are no large blood vessels, only a few muscle fibers, and, except in cases of extreme obesity and advanced pregnancy, a small amount of fat. The most important feature, however, is that it contains red marrow of fairly uniform quantity and of low fat content extending from the suprasternal notch to just above the xiphoid.

Penetrability: The bone may be pierced with the ordinary spinal needle, although many elaborate needles and drills with various guards have been used. The use of a small hammer has been suggested, but this would probably induce too much psychic trauma.

Safety: Due to the width of the sternum and the density of its inner table, there is little danger of entering the mediastinum while making a sternal puncture. The largest neighboring blood vessels are the mammary vessels which are located laterally about 1 cm. beyond the bone.

The sternum has almost insignificant disadvantages. In children younger than three years, the islands of hematopoiesis are isolated, variable in size and location and difficult to find. In the adult, roentgenograms of the sternum are of no value in determining preferred sites of aspiration because of the shadows of adjacent hard and soft structures. Other bones, such as the rib and tibia, are adjacent to vital structures, or are too resistant to penetration, or contain too much yellow fatty marrow to be suitable for study.

TECHNIC

One hour prior to the sternal marrow aspiration, the patient is given a rapidly acting barbiturate. The patient is placed on a firm table with no pillow so as to expose the maximum area of the sternum. In hairy-chested individuals the skin over the sternum is shaved. The skin is prepared with weak tincture of iodine and alcohol. With aseptic technic an eye sheet is placed over the sternum with the opening at the desired site. The operator palpates the suprasternal notch to locate the midline, and confirms the location by feeling for the intercostal spaces with the fingers of the opposite hand. The area just below the angle of Louis is the most frequently used because of its conspicuous location. The operator makes an intradermal wheal in the midline with 1 per cent procaine. The procaine is slowly infiltrated into the sternal subcutaneous tissue and periosteum; the latter is very sensitive and requires more anesthetic and time. An area of about 1 cm. in diameter is infiltrated. Most cases require not more than 2 c.c. When the patient experiences no pain on moving the needle over the periosteum, the needle is withdrawn.

The spinal needle with its stylet *in situ* is gently forced through the skin and subcutaneous tissue to the periosteum. Since the bone cannot be anesthetized, the patient

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is told that he will experience a little pain. At this time the needle is supported at two levels: the lower, near the skin, is held with the index finger and the thumb of the left hand to keep the needle from bending, and to prevent the needle from penetrating too deeply or too rapidly; at the upper level the right index finger and thumb are used to twist the hub and push the point through the outer table of the sternum. A "give" comparable to that experienced on piercing the dura in spinal puncture is palpated when the marrow cavity is entered.

As soon as the needle point enters the marrow cavity, the stylet is removed, a 10 c.c. syringe is inserted, and 1 c.c. of the marrow is aspirated. This frequently produces pain and it is advisable to caution the patient not to move. The needle and syringe are removed as a unit, and the contents are gently expelled through the needle into a bottle containing 2 mg. of a previously dried mixture of potassium and ammonium oxalate (Heller and Paul anticoagulant). The bottle is rotated for three minutes to prevent coagulation. Simultaneously, five slides are made from the marrow in the needle. In order to prevent shrinking due to osmosis, the slides are dried rapidly by fanning. From the beginning of aspiration to the deposition of the marrow into the bottle no more than 45 seconds should elapse because of the rapid coagulation of bone marrow.

Light pressure must be applied to the site of aspiration for five minutes after removal of the needle; otherwise there may be a painful hematoma in the subcutaneous tissue of periosteum. No dressing is needed. Within twenty minutes a drop of serum frequently oozes from the skin.

LABORATORY PROCEDURES

In the laboratory the bottle is rotated to re-mix its contents well. With a white diluting pipette marrow is drawn to the 1 mark and then diluted with 5 per cent acetic acid containing methylene blue (1 c.c. of 1 per cent methylene blue to 9 c.c of 5 per cent acetic acid). The acetic acid hemolyzes non-nucleated erythrocytes and leaves the immature erythroid elements as well as the other myeloid cells clearly defined and conspicuous. The total nucleated elements (T. N. E.) can be estimated by charging an ordinary Levy counting chamber and noting the number of nucleated elements present in five of the central squares (that is, as for the red count). Formula for T. N. E:

$$\text{T.N.E.} = \frac{\text{Number of cells counted} \times \text{Dilution (10)}}{\text{Area (0.02)}}$$

The megakaryocytes are counted from the fluid remaining in the pipette. After a thorough re-mixing, the fluid is placed in a Fuchs-Rosenthal counting chamber. This counter has a volume of 3.2 cu. mm. and is divided into 256 squares; therefore the following formula is used:

$$\text{Megakyocytes} = \frac{\text{Number of megakaryocytes counted} \times \text{Dilution (10)}}{\text{Area (3.2)}}$$

The undiluted marrow is next placed in a dry Wintrobe hematocrit tube and centrifuged for five to seven minutes (depending on layer formation) at 2,000 RPM. The marrow divides itself into four layers: (1) a layer of fat which is a pale red or yellow; (2) a layer of clear plasma; (3) the most important layer, composed mostly of nucleated cells and a few erythrocytes (sometimes called the buffy coat); (4) a layer composed of mature red cells and reticulocytes.

When the line of demarcation between the nucleated cell layer and the red cells is not distinct an additional minute of centrifugation will greatly aid separation.

Smears are made from the buffy layer. This concentration of nucleated cells is especially useful in cases of hypoplastic or aplastic anemia.

All slides are stained with Wright's stain. Staining time after dilution is double that used in peripheral blood staining since there is more nuclear material to absorb the stain.

The thin portion of the slide is examined with the high dry lens to note the frequency of the nucleated cells; this also gives a suggestion of the activity of the marrow. Large cells like the megakaryocytes are readily observed. With the oil immersion lens the ratio and stages of development of

TABLE 1
STERNAL MARROW FINDINGS IN HEALTH AND DISEASE

Diagnosis	Total nucleated elements per c. mm. in thousands	Percentage Distribution				Megakaryocytes per cu. mm.
		Fat	Plasma	Buffy layer	Red cell layer	
Normal male	87.0	3.0	43.0	7.0	43.0	22
Stem cell leukemia	150.5	0.0	45.5	22.5	32.0	6
Sickle cell disease	207.5	0.0	54.0	20.0	26.0	46
Pernicious anemia	47.5	1.7	74.5	6.3	17.5	50

TABLE 2
STERNAL MARROW IN HEALTH AND DISEASE

GRANULOCYTES

Diagnosis	Myeloblasts	Leukoblast	Promyelocytes	Neutrophilic myelocytes	Neutrophilic metamyelocyte	Neutrophiles	Eosinophilic myelocytes	Eosinophilic metamyelocytes	Eosinophiles	Basophilic myelocytes	Basophilic metamyelocytes	Basophiles	Heterophiles	Granulocyte mitosis
Normal male	0.3	1.7	3.9	8.1	12.3	19.3	0.9	1.0	1.9	0.1	0.1	0.1	0.3	0.1
Stem cell leukemia	0.0	0.0	0.0	0.2	0.3	3.6	0.0	0.1	0.3	0.0	0.1	0.1	0.0	0.6
Sickle cell anemia	1.7	0.6	1.4	3.7	5.5	8.7	0.1	0.2	1.1	0.2	0.0	0.0	0.3	0.1
Pernicious anemia	0.4	1.5	2.6	8.3	6.6	22.8	0.1	0.1	0.5	0.2	0.2	0.0	0.2	0.1

TABLE 3
STERNAL MARROW IN HEALTH AND DISEASE

ERYTHROID SERIES

Diagnosis	Pronormoblasts	Basophilic normoblasts	Polychromatic normoblasts	Orthochromatic normoblasts	Promegaloblasts	Basophilic megaloblasts	Polychromatic megaloblasts	Orthochromatic megaloblasts	Erythroid mitosis
Normal male	0.6	2.1	6.9	16.7	0.0	0.0	0.0	0.0	0.7
Stem cell leukemia	0.1	0.3	0.9	2.9	0.0	0.0	0.0	0.0	0.0
Sickle cell anemia	2.4	6.9	11.9	34.2	0.0	0.0	0.0	0.0	1.4
Pernicious anemia	2.9	4.3	4.1	4.1	4.8	2.4	2.0	1.2	0.8

TABLE 4
STERNAL MARROW IN HEALTH AND DISEASE

AGRANULOCYTES

Diagnosis	Monocytes	Lymphocytes	Immature lymphocytes	Plasma cells	Stem cells	Reticulo-endothelial cells	Damaged cells	Granulocytic lymphocytic erythroid ratio	Peroxidase positive Peroxidase negative
Normal male	0.3	9.8	0.3	0.1	0.0	0.3	12.1	5:1:3	1.5:1
Stem cell leukemia	0.0	3.5	0.0	0.1	77.1	0.1	9.7	*	1:20
Sickle cell anemia	0.0	11.3	0.1	0.6	0.0	0.4	7.2	2:1:6	1:4
Pernicious anemia	0.1	13.5	0.2	0.1	0.0	0.8	15.1	4:1:2	1:1

*Since stem cells are so immature and cannot be positively identified they cannot be classified as myeloblasts or lymphocytes.

the granulocytic, erythroid and lymphoid cells are studied. The cytologic details may be observed but unfortunately the details of marrow architecture are lost. If time is available a differential count should be done using 500 to 1,000 cells.

DISCUSSION

Fourteen normal individuals were studied at Charity Hospital to determine the normal marrow findings. The individuals studied were doctors, medical students, and technicians whose ages ranged from 17 to 27 years.

In ten normal males the total nucleated elements varied between 43,000 and 105,000 per cu. mm., and in four normal females the T. N. E. ranged between 30,000 and 87,000 per cu. mm.

The megakaryocytes are large cells measuring approximately 30 micra or more. They may be multinucleated or may possess a multilobular nucleus. There is a large amount of cytoplasm which frequently is surrounded by a clear halo. The total number of megakarocytes in normal individuals varies from 6 to 22 per cu. mm.; further work on normal counts is being done and will be reported later.

The percentage proportion of the centifuged marrow as derived from the 14 normal individuals is as follows:

	Per cent
Fat	1 - 3
Plasma	40 - 54
Nucleated elements	3 - 11
Red cells	40 - 51

Tables 1 to 4 exhibit the recommended form for recording the data. The data are derived from one normal individual and typical cases of three blood dycrasias. All differential counts were made by counting 1,000 cells.

For the beginner the peroxidase stain will be of great aid in cell identification. Cells of the erythroid and lymphoid series are peroxidase negative, and while the myeloblasts and leukoblasts are frequently negative, positive granules are found occasionally. Their fine nuclear pattern will make the final differentiation. Promyelocytes have many peroxidase positive granules and the remaining cells of the granulocytic series are definitely peroxidase positive. Pronormoblasts and promegaloblasts are peroxidase negative with a fine nuclear pattern and can be differentiated from the myeloblast by their deep blue cytoplasm. The promegaloblast has a greater nucleocytoplasmic ratio than the pronormoblast.

Some authors distinguish between large, medium sized, and small lymphocytes; such a classification is time consuming and does not appear to aid in diagnosis or prognosis. We do not attempt to distinguish on morphological grounds monoblasts or lymphoblasts from myeloblasts, since in our experience the various primitive white cell precursors that have been described by various authors are not sufficiently clear-cut or constant to permit their recognition in smears with any confidence.

SUMMARY

The sternum is the site of choice for bone marrow aspiration because of its accessibility, penetrability and safety. The marrow is removed with a spinal needle and is mixed with Heller's oxalate. The total nucleated elements and megakaryoctyes are counted from this oxalated marrow. Smears made from the untreated marrow and from the buffy layer are stained with Wright's stain. Tables are furnished citing the sternal marrow findings in a normal individual and in three types of blood dyscrasia.

THE "BORROWING-LENDING" HEMODYNAMIC PHENOMENON (HEMOMETAKINESIA) AND ITS THERAPEUTIC APPLICATION IN PERIPHERAL VASCULAR DISTURBANCES*

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While it has long been known that the

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volume of organs undergoes spontaneous and even rhythmic variations, it was not until the more refined technics of plethsmography became available that a better understanding of these phenomena were obtained. With such technics,[1] studies of a more precise and quantitative nature could be made of these fluctuations in volume in peripheral parts such as the fingers, toes, and pinnae of the ears. These variations in volume have been attributed essentially to changes in the volume of the vascular bed of the part, and thus reflect variations in behavior of the local circulation. As a result of these studies, much valuable information has been obtained concerning the hemodynamics of the peripheral circulation under normal resting conditions, in diseased states, and in response to external and internal stimuli. Although there may be many factors which remain unknown or poorly understood, certain facts have been established which suggest a more rational approach to certain clinical conditions. For these reasons, it seems desirable to present a consideration of these facts and their application to clinical conditions characterized by a disturbance in peripheral circulation.

The plethysmogram may be regarded essentially as an ordinary type of Cartesian coordinate, with the volume represented on the ordinate and time on the abscissa. It is, therefore, a record of the changes in volume of the part enclosed in the extremity cup in relation to time. The recorded volume changes are the algebraic summation of many volume changes, which are taking place in all portions of the enclosed part. Essentially, however, the recorded changes in volume of the part represent variations in the volume of the vascular bed of the part.

By means of plethysmographic studies of this kind it has been shown that the vascular bed in such peripheral parts as the pinnae, the fingers, and the toes undergo spontaneous variations in volume.[1, 2] In the resting individual in a comfortable environment, at least five types of rhythmic changes in volume have been observed. They have been described in detail elsewhere and have been given the following designations: (1) *pulse* deflections, (2) *respiratory* deflections, (3) *alpha* deflections, (4) *beta* deflections, and (5) *gamma* deflections. They range in volume from less than 0.1 to 350 or more cubic millimeters per 5 c. c. of part (fig. 1).

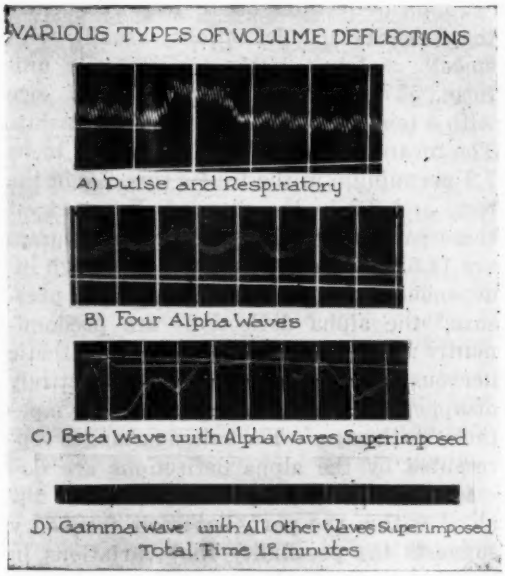
VARIOUS TYPES OF VOLUME DEFLECTIONS
A) Pulse and Respiratory
B) Four Alpha Waves
C) Beta Wave with Alpha Waves Superimposed
D) Gamma Wave with All Other Waves Superimposed
Total Time 12 minutes

Fig. 1. A. Plethysmogram of a normal subject showing pulse and respiratory deflections. B. Four unusually regular alpha waves. C. Beta wave with alpha waves superimposed. D. A short gamma wave with other waves superimposed. Time, 12 minutes.

The pulse deflections which represent the changes in volume of the part brought about by the blood delivered into the part with each heart beat vary considerably in volume, with mean values of 6.9 cubic millimeters in the finger tips, 4.0 in the toe tips, and 4.1 in the pinnae per 5 c. c. of part.*[2] The changes in volume are probably due to variations in content of all components of the local circulatory bed; arteries, arterioles, capillaries, venules and veins.

The respiratory deflections represent variations in volume occurring with the normal respiratory cycle and their volumes vary from less than 0.1 to 5 cubic millime-

*Hereafter all volume changes are expressed per 5 c.c. of part.

ters.[2] They reflect variations in venous return to the heart brought about by respiration. In addition the changes in heart rate and changes in the heart output associated with respiration contribute considerably to the development of these variations in volume.

Alpha deflections occur less frequently than respiratory deflections, usually with smooth contours, but not necessarily uniform. They vary in frequency and size with a tendency for an inverse relationship. The mean frequency has been found to be 7.9 per minute in the finger tips, 7.7 in the toes, and 8.6 in the pinnae.[2] For each of these parts respectively, the mean volumes are 14.5, 7.1, and 6.6 cu. mm.[2] Although independent of variations in arterial pressure,[3] the alpha deflections are predominantly under the control of the sympathetic nervous system, for they almost entirely disappear following interruption of sympathetic pathways.[2] The volume changes represented by the alpha deflections are due essentially to variation in volume of the blood within the part, but their frequency suggests the possibility that variations in lymph volume may be a contributing factor. Experimental studies in animals have shown contractions of the lymphatics to be responsible for much of the flow of lymph.[4, 5, 6] If these lymphatic contractions occur in man and at the same frequency as observed in animals they might contribute to the volume changes.

Beta deflections represent larger and more slowly developing changes in volume upon which alpha waves are superimposed. Their frequency is one to two per minute; the volume varies from 5 to 60 cu. mm.[2] Although they may be irregular in rhythm and volume they tend to vary concordantly in the fingers, toes and pinnae. These deflections are also considered to represent primarily volume changes in the vascular bed, but like the alpha deflections, a significant part of the volume change may be due to variations in lymph volume. Changes in inter- and intracellular volume may also contribute to these deflections.

The gamma deflections are due to still more slowly developing changes in volume upon which the beta waves are superimposed. Their frequency varies from one to eight an hour and their magnitude from 50 to 350 cu. mm.[2] The gamma deflections are predominantly the result of changes in volume of the vascular bed of the part, although variations in lymph volume probably contribute significantly to the volume change. It would appear that the gamma volume deflections are more likely concerned with large and usually relatively slow shifts in blood volume from one part of the body to another.

Under certain circumstances considerable variations may be found in the plethysmogram of normal individuals. Because these changes may resemble those found in diseased states, it is desirable to review them briefly. Among the factors which may produce these changes are the psychic state of the individual, the temperature of the environment, the relation of the part to heart level, and the presence or absence of intact sympathetic pathways to the part.

The psychic state of the individual has a profound influence upon the pulse and alpha deflections. For example, fear, anxiety, or tenseness on the part of the individual tend to produce a diminution in the pulse and alpha deflections and an increase in the rate of the pulse deflections.[7, 8] This is apparently due to an increased sympathetic activity associated with the psychic disturbance, causing an increase in vasomotor tone. The character of the spontaneous volume deflections may thus serve as a good reflection of the psychic state of the individual.

The environmental temperature may also produce marked changes in the spontaneous volume deflections. For example, chilling of the subject, either by local application or general environmental cold produces vasoconstriction and a consequent reduction in the volume of the pulse and alpha deflections and a decrease in the total volume of the part (negative gamma deflection). In general the degree of vasoconstriction is proportionate to the degree of chilling. On the other hand, a warm environment has

the reverse effect, producing a vasodilatation and a consequent increase in the volume of the pulse deflections. As the part warms, the alpha deflections are increased. Later, after full vasodilatation there is a decrease in the alpha deflections. The positive gamma deflection which follows warming the subject indicates an over-all increase in the volume of the part. As has been indicated previously, these reactions serve as a good test for organic occlusive arterial and arteriolar disease.[1] In the presence of these conditions, such as thrombo-angiitis obliterans or obliterating arteriosclerotic endarteritis, these vasodilating responses are impaired or absent. The effort to produce vasodilatation in the fingers or toe tips by the application of heat to another extremity tests the patency of the blood vessels to the part under investigation as well as the neurovascular mechanism of the part.

The position of the part in relation to heart level also affects these spontaneous volume deflections. Placing the part below heart level, for example, produces a decrease in the volume of pulse and alpha deflections. The pooling of blood and lymph in the dependent part as a result of gravity causes a positive gamma deflection. Two factors, either alone or in combination, have been suggested as a means of explaining this phenomenon. The first is arteriolar vasoconstriction and the second, which is more likely, is distention of the vessels secondary to increased venous pressure, with a diminution in further distensibility of the vessel wall.[9] Elevation of the part above heart level produces the reverse effect. There is pronounced increase in the volume of the pulse and alpha deflections and as a consequence of the draining out of the blood within the part, a negative gamma deflection. For these reasons, the part should be kept at or near heart level for standard recordings.

The spontaneous volume deflections are greatly affected by the state of the sympathetic pathways to the part. Thus, within a few minutes after the regional sympathetic nerves or ganglia have been blocked by infiltration with 1 per cent procaine hydrochloride solution, there is a marked increase in the volume of the pulse deflections and virtual disappearance of the alpha deflections. The engorgement of the vascular bed within the part following the sympathetic block also produces a large positive gamma deflection. The procedure of sympathetic block is another significant diagnostic test,[10, 11] for by this means it is readily possible to determine the degree of the normal, or, in certain conditions, the abnormal sympathetic vasoconstrictor tone and the extent of vasodilatation or the general order of the amount of increase in vascularity of the part that can be achieved by interruption of these pathways.

THEORETICAL CONSIDERATIONS OF THE "BORROWING-LENDING" HEMODYNAMIC PHENOMENON (HEMOMETAKINESIA)

In addition to the spontaneous variations in the volume of the vascular bed of such peripheral parts as the fingers, toes, and pinnae, which are represented on the plethysmogram by the alpha, beta, and gamma deflections, similar variations in the volume of certain internal organs have been demonstrated. For example, Richards and Schmidt[12] observed an intermittent blood flow through kidney glomeruli and Krogh[13] made similar observations in the tongue and skeletal muscles of the frog. The irregularities in the blood flow in the rabbit's ear were also reported by Grant.[14] Further experimental observations by Zweifach[15] and Chambers and Zweifach[16] on the circulation in various organs have demonstrated clearly the intermittency of blood flow in different parts of the body, a phenomenon which they termed vasomotion. The spontaneous variations of the splenic volume in the dog, reported by several observers,[17-21] are of a magnitude and frequency comparable to those of the beta and gamma deflections described above.

It is readily evident from these considerations that spontaneous variations in the vascular bed of different parts of the body are constantly occurring. These changes in the volume of blood in widely separated parts of the body may be rhythmic, concordant, or discordant. They may be pro-

duced by intrinsic factors, which are not yet well understood, or may be influenced by various internal and external stimuli. They indicate a continuous shifting back and forth of the blood from one part of the body to another to meet local requirements.[22] They suggest a well regulated mechanism, with definite order and significance, which appears to be concerned with certain vital functions such as proper thermal regulation, nutrition, repair, and other important physiologic adjustments.

It is conceivable that at least a part of this significance lies in the attempt by the body mechanism to utilize its total blood volume in the most efficient manner. Obviously the total volume of blood in the body at any one time is too small to meet the maximum demand of all the tissues at the same time, should a disease state arise to precipitate such a situation. The total blood volume, on the other hand, is more than sufficient to meet urgent demands for large quantities of blood in isolated parts. While great variations can take place in the vascular bed within a relatively short period of time, especially in isolated parts of the body, the total blood volume remains relatively constant under ordinary conditions. For example, the vascular volume of a finger or toe can be observed to double in size within a matter of minutes. To fill this augmented vascular bed with blood, however, obviously does not require doubling the total blood volume nor is there reason to believe that there exists in the body a special reservoir of blood for this purpose. Yet, it must have come from some source and the obvious explanation is that it was taken from the vascular bed of other parts of the body. In other words, the total blood volume need not be changed by even one cubic centimeter. The only change required to achieve this purpose is an adjustment in the vascular bed. By increasing the volume of the vascular bed in one part of the body and decreasing it by the same amount in other parts, the volume of blood in the former area is increased at the expense of the latter without any variations in the total volume of the blood.

To illustrate this "borrowing-lending" phenomenon (hemometakinesia), the following observations are presented. These data have been selected from many similar experiences in normal individuals as well as patients with various forms of peripheral vascular disease. The results of these studies have been invariably consistent, regardless of the methods employed to determine and record variations in blood flow. Our experiences include particularly the use of thermometric and phlethysmographic methods of study. These observations are routinely made under controlled atmospheric conditions in a room constructed to reduce psychic disturbances. The subjects rest in bed for a time sufficient to permit stabilization of the vascular system. Plethysmograms are obtained for the distal phalanges of the fingers and toes, and for the pinnae of the ears. The index finger and the second toe are most commonly employed. Thermograms are obtained for these parts as well as others distributed bilaterally and symmetrically over the body, a total of twenty areas usually being observed. The plethysmographic and thermometric observations are usually made simultaneously. The response to various measures, such as interruption of sympathetic innervation, reactive hyperemia, drugs, environmental and local temperature changes, and pyschic and neurogenic factors are observed as reflections in the plethysmograms and thermograms.

CASE NO. 1

G. I., a white male 17 years of age, was examined because of marked hyperhidrosis. Plethysmographic and thermometric studies were made after the patient had rested in bed in the observation room (room temperature 78° F. and relative humidity 46 per cent) for 50 minutes. Skin temperature determinations were made bilaterally on the third toe, the dorsum of the foot, the mid-pretibial area, the knee, the mid-thigh and the chest. The plethysmographic determinations were made for the distal phalanges of the right index finger and for the second toe bilaterally. After the patient had reached a state of stabilization 500 mgm. of tetraethyl ammonium chloride[23] were injected intravenously. Then after sufficient time had elapsed for the reaction to subside a left lumbar sympathetic nerve block was performed with 1 per cent procaine hydrochloride solution. The vascu-

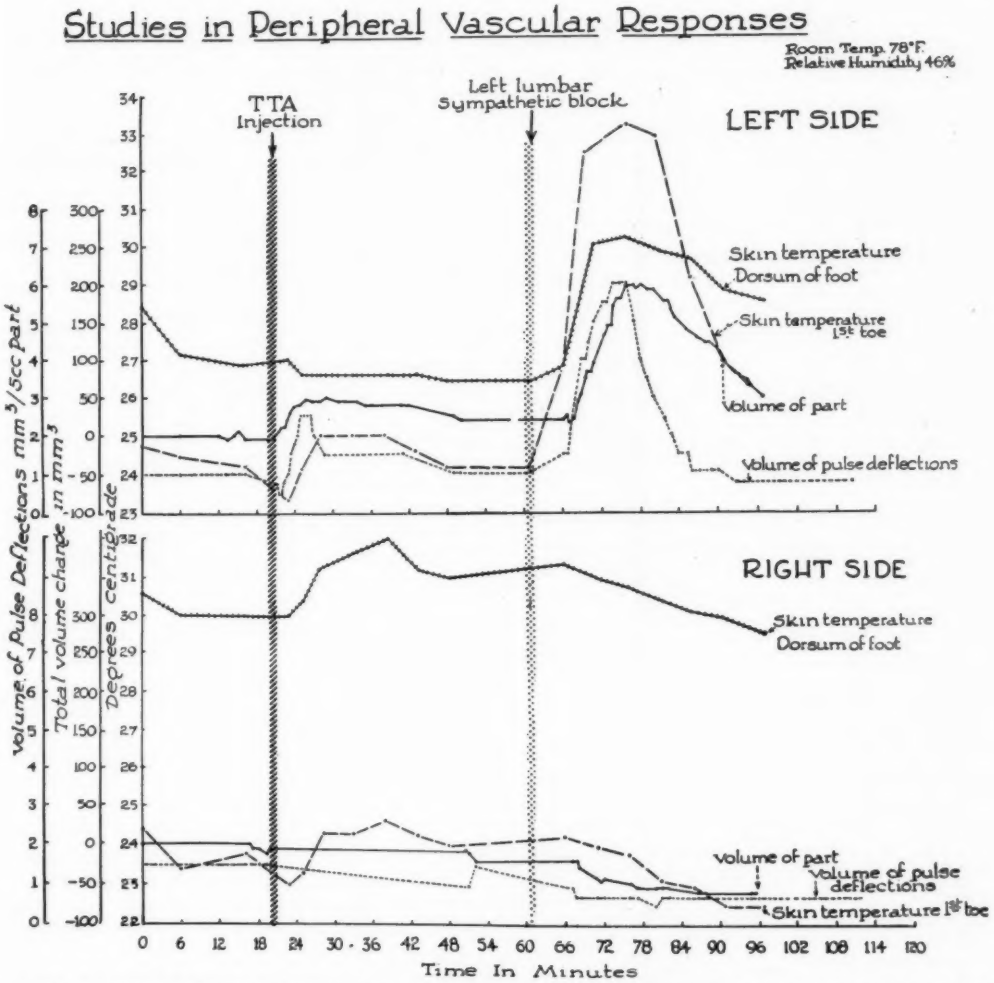

Fig. 2. Graph showing thermometric and plethysmographic response following injection of tetraethyl ammonium chloride (T.T.A.) and left lumbar sympathetic block. See text for details.

lar phenomena resulting from these procedures are shown in figure 2.

Initially the patient was stabilized in a state of moderate vasoconstriction as evidenced by: (1) small pulse deflections; (2) small fluctuations in the total volume of the part; (3) relatively low skin temperature of the toes.

The procedure of intravenous injection of tetraethyl ammonium chloride resulted in a further vasoconstriction as evidenced by a decrease in the volume of pulse deflections. Following the injection of tetraethyl ammonium chloride there was a slight vasodilatation shown by an increase in the volume of pulse deflections, an increase in the total volume of the part and a rise in skin temperature. These reactions were slight. There were skin temperature changes on the right side of the same magnitude as on the left but no determinations of pulse deflections and total volume were made in the right toe during this period.

Within a few minutes after the left lumbar sympathetic block was done the following phenomena were observed:

1. A rapid elevation of the skin temperature of the left leg with marked flushing and an absence of sweating.
2. An increase in the volume of the pulse deflections of the left second toe.
3. An increase in the total volume of the left second toe.
4. A fall in the skin temperature of the right leg.
5. A decrease in the volume of the pulse deflections of the right second toe and index finger.
6. A decrease in the total volume of the right second toe and index finger.

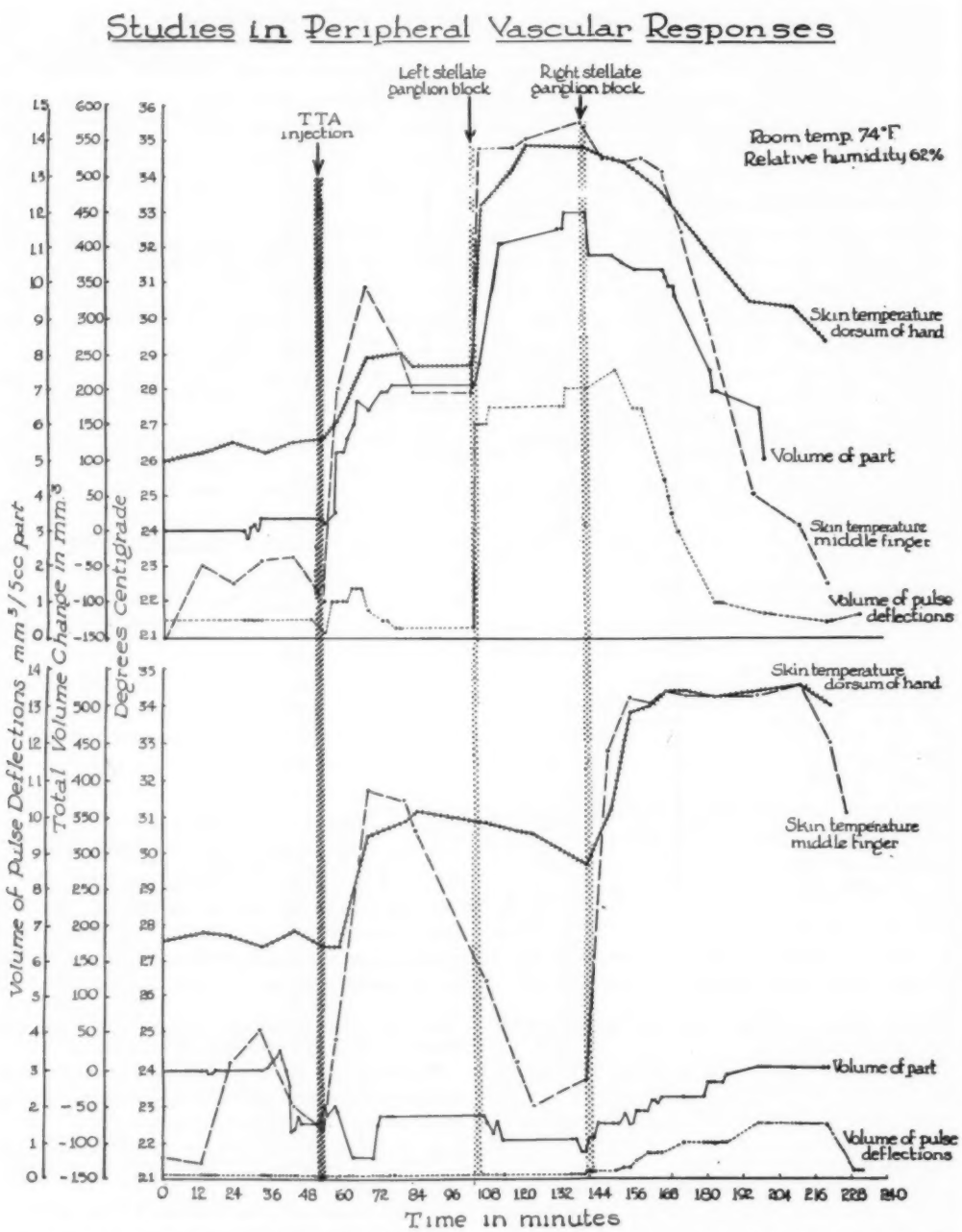

Fig. 3. Graph showing thermometric and plethysmographic response to injection of tetraethyl ammonium chloride and left and right stellate ganglion block. The upper half is the left side, the lower half is the right. See text for details.

These changes indicate a vasodilatation in the left leg. There was an increase in the volume of blood and rate of blood flow to this leg which occurred at the expense of the vascular bed of the remainder of the body. That blood was borrowed from the remainder of the body is evidenced by the fall in skin temperatures, the fall in total volume and volume of pulse deflections in all other areas. The magnitude of changes observed in the areas which were concerned with "lending" were small,

indicating that the remainder of the vascular bed and not any one single area is involved. The normal physiologic adjustments are such that no one organ or tissue is called upon to give up its blood to an extent which would produce injury as a result of ischemia. The previous state of vasoconstriction or vasodilatation will determine in large part the degree of lending possible. As demonstrated in this subject, a part which is already in a state of vasoconstriction (the right toe) can not give up as much blood as would be expected of a part in mid or full vasodilatation.

CASE NO. 2

A.M., a white male aged 59, suffered from arteriosclerosis and Raynaud's phenomenon. After resting in bed for 60 minutes in an environmental temperature of 74° F. and 64 per cent relative humidity the following studies were made; these included a measurement of skin temperatures of the first toe, dorsum of the foot, mid-pretibial area, knee, mid-thigh, chest, deltoid region, forearm, dorsum of the hand and middle finger bilaterally as well as plethysmographic studies of the right second toe, the left index finger and the right fourth finger. The latter was selected instead of the right index finger because of the more severe cyanosis which the fourth finger presented.

After a state of stabilization had been reached, 5 c.c. (500 mgm.) of tetraethyl ammonium chloride were given intravenously. Fifty minutes later a left stellate ganglion block was performed with 1 per cent procaine. Then after another 35 minutes the right stellate ganglion was blocked in the same manner.

Initially the right ring finger was cyanotic and the entire right hand presented a mottled cyanotic appearance. The left index finger was cyanotic but not as marked as the right fourth finger. The initial skin temperatures were low on both sides and the pulse deflections were small. Following the injection of tetraethyl ammonium chloride the skin temperatures of both hands increased significantly. There was an increase in the total volume of the left index finger and a small increase in the pulse deflections. The right finger did not show pulse or volume changes. Coincident with the increase in temperature the skin color improved but did not reach normal. Cyanosis returned before the left stellate ganglion block was performed.

Immediately following the left stellate ganglion block the following phenomena were observed (fig. 3):

1. An immediate rise in skin temperature of the left arm and hand with the most pronounced changes distally.

2. A flushing of the left arm and hand with a return of normal skin color.

3. A pronounced increase in the pulse deflections.

4. A marked increase in the total volume of left index finger.

5. A decrease in the skin temperature of the right arm and hand.

6. A decrease in volume of the right finger.

7. An increase in the degree of cyanosis of the right finger.

Immediately after the right stellate ganglion block the following changes were noted:

1. A marked increase in skin temperature, again most pronounced distally.

2. Disappearance of the cyanotic color.

3. A significant increase in the volume of the pulse deflections.

4. An increase in the total volume of the right finger.

5. A decrease in skin temperature of the left arm and hand.

6. A decrease in the total volume and the volume of the pulse deflections of the left finger.

7. Reappearance of a mottled cyanotic skin color in the left hand.

Terminally, the volume of the pulse deflections decreased in the right finger and the cyanosis reappeared.

The changes observed in this subject indicate vasodilatation following injection of tetraethyl ammonium chloride. Following the left stellate ganglion block there was an increase in the volume of blood and the rate of blood flow in the left arm. This increase in the left side is indicated by the rise in skin temperature, total volume and volume of the pulse deflections. That this occasioned a "lending" from the right arm is evidenced by a decrease in skin temperature and total volume. The volume of the pulse deflections on the right was very small and could not be diminished any further. The preexisting state of the right finger precluded its lending but a very small amount of blood without danger of ischemia. The increase in cyanosis could be interpreted as a deleterious effect—an actual deprivation below the very minimal needs for viability of the tissues.

Immediately after the right stellate ganglion block there was vasodilatation in the right upper extremity. This is shown by the occurrence of the same changes demonstrated in the left arm when the left stellate ganglion was blocked. The cyanosis on the right side disappeared. However, when the shift of blood began toward the right arm the left was in a state of marked vasodilatation. It, therefore, was able to "lend" more blood than was possible for the right arm previously. That this is precisely what occurred is shown by the marked decreases in skin temperature, total volume and volume of pulse deflections in the left side as compared to the small changes which occurred previously with a small "loan" from the right to the left arm.

DISCUSSION

From the foregoing considerations, it seems evident that there is a continuous shifting back and forth of blood from one part of the body to another. It is suggestive of a borrowing and lending of blood, a phenomenon for which we should like to suggest the term "hemometakinesia." This borrowing and lending of blood from tissue to tissue, to meet variations in requirements is indicative of a well regulated mechanism which permits the body to utilize its limited total blood volume in the most efficient manner. The essence of this mechanism lies in the control and regulation of the vascular bed. By this means, adjustments in the vascular bed can be made to permit an increase in the volume of blood in one part of the body with a corresponding simultaneous decrease in the volume of blood in other parts of the body. Thus, the blood flow to different parts of the body may be varied considerably without affecting the total blood volume or cardiac output.

It is well to point out that when blood is borrowed from any organ, this blood is removed from all blood vessels of the organ. A larger part, however, must come from the large vessels within the organ, such as the venous reservoirs. The relative contributions made by the small vessels are unknown. From the studies of others[24] the small vessels would be expected to contribute a relatively small portion.

In approaching therapy designed to increase the local circulation to a part, such as in peripheral vascular disease, the application of similar principles of hemodynamics appears rational. These conditions are characterized by a disturbance or an actual diminution in the normal amount of circulating blood to a part. Effective therapy is aimed at improvement in the circulation or increase in blood supply to the part. According to the principles of hemodynamics described above, it would not be advisable even were it possible to achieve this purpose by therapeutic measures designed to produce dilatation of the entire vascular bed, when a diseased state is localized to a single peripheral part. Nonetheless, medical literature is replete with articles reporting studies and claims for agents directed at local therapy through the production of generalized vasodilatation. Should such an agent, with the ability to produce maximum generalized vasodilitation, exist and be employed, the reaction would be shock-like, completely defeating the original purpose of increasing the blood supply to a local part. This becomes readily evident when it is realized that should the entire vascular bed suddenly increase greatly in volume, a great disproportion would immediately occur between the volume of the vascular bed and the available volume of blood. With a relatively fixed total blood volume, it would be impossible to increase the circulating blood volume in any one single part or tissue if there were a comparable increase in the volume of the entire vascular system. Furthermore, if the volume of the entire vascular bed increased suddenly, the drop in intravascular pressure or blood pressure would be so great, that the blood flow in a localized part would be impaired rather than improved. It seems evident, therefore, that therapeutic measures designed to provide the maximum increase in the blood supply to a local part, through the medium of a generalized vasodilatation, is theoretically unsound.

On the other hand, therapeutic measures having as their purpose the production of local vasodilatation limited essentially to the part in need of more blood, is theoretically sound and potentially effective. In our experience, this is best accomplished by sympathetic denervation of the affected part. This therapeutic approach conforms with the principles of hemodynamics described above and provides the maximum local vasodilatation which is possible in a given case without reducing the arterial blood pressure or producing serious systemic disturbances in hemodynamics by the sudden creation of a disproportion between the total volume of the vascular bed and the total volume of blood. Theoretically there should be, and actually, as is illustrated by the case described above, there has been found to be a definite and significant in-

crease in blood supply to the diseased part by this form of therapy. In fact, we have yet to find a general vasodilator agent, which could produce in a local part, such as the toes, fingers, foot, hand, or an entire extremity, vasodilatation equal in degree or duration to that produced by sympathetic denervation of the part.

SUMMARY

Although some phenomena of the peripheral circulatory reactions may not be completely understood, the facts which have been established indicate that there is a continuous shifting of blood from one part of the body to another. This "borrowing and lending" indicates a well regulated mechanism which permits the body to meet the marked variations in local requirement of blood with a limited total blood volume. The underlying principle is a control of the vascular bed which permits an increase in the volume of blood in one area with a simultaneous decrease in other areas. In this manner the total blood volume is unaltered.

For this "borrowing-lending" mechanism the term *hemometakinesia* is proposed.

It is suggested that this hemodynamic principle is applicable in the management of peripheral vascular diseases in which the aim of therapy is an increase in the blood supply to the part. Measures which are directed toward improvement of the local circulation by the production of generalized vasodilatation are of doubtful value and not in conformity with the natural physiologic mechanism. However, sympathetic denervation, the most effective method of increasing local blood supply, is in complete conformity with the concept of hemometakinesia.

Two cases are presented to illustrate the "borrowing-lending" phenomenon and to demonstrate the value of sympathetic denervation as a therapeutic measure in peripheral vascular disease.

REFERENCES

1. Burch, G. E.: A new sensitive portable plethysmograph, Am. Heart J., 33:1, 1947.

2. Burch, G. E., Cohn, A. E., and Neumann, C.: A study by quantitative methods of the spontaneous variations in volume of the finger tip, toe tip, and postero-superior portion of the pinna of resting normal white adults, Am. J. Physiol., 136:433, 1942.

3. Neumann, C.: A study of the effect of spontaneous variations in blood pressure upon the spontaneous variations in the volume of the finger tip, Am. J. Physiol., 138:618, 1943.

4. Webb, R. L., and Nicoll, P. A.: Behavior of lymphatic vessels in the living bat, Anat. Rec., 88:351, 1944.

5. McMaster, Philip D.: An inquiry into the structural conditions affecting fluid transport in the interstitial tissue of the skin, J. Exper. Med., 74:9, 1941.

6. McMaster, Philip D.: Conditions in the skin influencing interstitial fluid movement, lymph formation, and lymph flow, Ann. New York Acad. Sci., 46:679, 1946.

7. Burch, G. E., Cohn, A. E., and Neumann, C.: Reactivity of intact blood vessels of the fingers and toes to sensory stimuli in normal resting adults, in patients with hypertension, and in senile subjects, J. Clin. Invest., 21:655, 1942.

8. Neumann, C., Cohn, A. E., and Burch, G. E.: A study of the influence of the character of an examining room on the peripheral blood vessels of normal, hypertensive and senile subjects, J. Clin. Invest., 21:651, 1942.

9. Burch, G. E., DeBakey, M., and Sodeman, W. A.: Effect of venous pressure on volume pulsation, Proc. Soc. Exper. Biol. & Med., 42:858, 1939.

10. Ochsner, Alton, and DeBakey, Michael: Peripheral vascular disease. Classification and therapeusis based upon physio-pathologic alterations, Internat. Clin., 3:1, 1939.

11. Ochsner, Alton, and DeBakey, Michael: Peripheral vascular disease. A critical survey of its conservative and radical treatment, Surg., Gynec. & Obstet., 70:1058, 1940.

12. Richards, A. N., and Schmidt, C. F.: A description of the glomerular circulation in the frog's kidney and observations concerning the action of adrenalin and various other substances upon it, Am. J. Physiol., 71:178, 1924-25.

13. Krogh, A.: The Anatomy and Physiology of Capillaries. Rev. ed. Yale University Press, New Haven, 1929.

14. Grant, R. T.: Observations on direct communications between arteries and veins in the rabbit's ear, Heart, 15:281, 1929-31.

15. Zweifach, B. W.: The character and distribution of the blood capillaries, Anat. Rec., 73:475, 1939.

16. Chambers, Robert, and Zweifach, B. W.: Functional activity of the blood capillary bed, with special reference to visceral tissue, Ann. New York Acad. Sci., 46:683, 1946.

17. Schafer, E. A., and Moore, B.: On the contractility and innervation of the spleen, J. Physiol., 20:1, 1896.

18. Hargis, E. H., and Mann, F. C.: A plethysmographic study of the changes in the volume of the spleen in the intact animal, Am. J. Physiol., 74:180, 1925.

19. Barcroft, J., Khanna, L. C. (Lahore), and Nisimaru, Y. (Okayama): Rhythmical contraction of the spleen, J. Physiol., 74:294, 1932.

20. Barcroft, J., Nisimaru, Y. (Okayama), and Puri, S. R. (Lahore): The Action of the splanchnic nerves on the spleen, J. Physiol., 74:321, 1932.

21. Grindlay, J. H., Herrick, J. F., and Blades, E. J.: Rhythmicity of the spleen in relation to blood flow, Am. J. Physiol., 127:119, 1939.

22. Burch, G. E.: Discussion of paper by McMaster, ref. no. 6.

23. Berry, R. L., Campbell, K. N., Lyons, R. H., Moe, G. K., and Sutler, M. R.: The use of tetraethylammonium in peripheral vascular disease and causalgic states, Surgery, 20:525 1946.

24. Gibson, J. S. II, Peacock, W. C., Seligman, A. M., and Sack, T.: Circulating red cell volume measured simultaneously by the radioactive iron and dye methods, J. Clin. Invest., 25:838, 1946.

THE TREATMENT OF UTERINE PROLAPSE*

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In the writings attributed to Hippocrates[1] in the fourth century B. C. is an excellent description of complete uterine prolapsus, in which he advises washing the parts with black wine, manual reposition, and seven days' rest in bed with the feet elevated. No mention is made, however, of the end results of this procedure.

Soranus,[2] of Ephesus, of the second century A. D., is our leading authority on the obstetrics and gynecology of the ancients. Even in that day, perineal bandages and pessaries were looked upon as time-honored methods of treatment. Soranus chides Euphyron for suspending his patients by the feet to a ladder for a period of 24 hours, and scores those who believed that the prolapsed uterus would retreat to its natural position if exposed to evil-smelling medicaments. He advocated manual replacement, binding the legs together, and, after several days' rest, use of a pessary of wood dipped in myrrh.

Little if any progress was made in the treatment of uterine prolapse during the middle ages, or in the Renaissance, for we find that during this time Rodericus a Castro[3] attacked the procidentia with a red hot iron to cause it to retreat into the vagina, while others bound lizards and mice to the uterus to frighten it into normal position. Paré[4] advised the use of a pessary consisting of a ball covered with leather, but does not mention surgery for the correction of the condition.

In 1702, Stephen Blancard[5] inferred surgical treatment of prolapse in his definition of "Procidentia Uteri" in The Physical Distionary:

"A relaxing of the Inner Tunick of the Womb, which falls through the Privities and was cut off by Physicians."

The first suggestion of a rational surgical approach to treatment of prolapsus is attributed to the French surgeon, Romaine Gerardin,[6] who, in 1823, proposed obliteration of the vaginal orifice by suture of the denuded labia. He did not, however, perform the operation. Eight years later (1831) Marshal Hall,[7] of London, proposed a rectangular denudation of the anterior vaginal wall followed by side-to-side suture of the denuded area in such a manner that the prolapsed uterus would be supported by the narrowed vagina. Heming[8] followed Hall's suggestion and performed the operation with success in 1931.

Shortly thereafter, Geddings[9] is credited with first combining perineorrhaphy with narrowing of the vaginal walls. Judging from the description of these early operations and from the accompanying illustrations, the operators depended almost entirely upon suture of the mucosa and skin. The deeper supportive structures were not repaired, and, in most instances, the operation served only to narrow the vaginal orifice, so that a ring pessary could be utilized. Important contributions were made to vaginal plastic surgery by Diffenbach, Velpeau, Marion Sims, Emmet and others during the middle of the nineteeth century. In 1877 Leon LeFort[10] advocated partial occlusion of the vagina by suturing together the denuded anterior and posterior vaginal walls. Almost simultaneously Neugebauer,[11] in Germany, devised much the same operation, which, with little modification, is now found useful in a definite but small group of cases.

In 1888, Donald,[12] of Manchester, England, then senior resident surgeon in St. Mary's Hospital, devised the combination of anterior and posterior colporrhaphy with amputation of the cervix, and called particular attention to the necessity of suture of the bases of the broad ligaments anterior to the cervix. This procedure was later modified by Fothergill[13] and more recently by Fletcher Shaw,[14] but with little change, the Manchester operation constitutes today the most effective treatment of the majority of cases of uterine prolapse.

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In the choice of treatment for uterine prolapse, patients must be individualized by consideration of many pertinent factors. Age, general condition of the patient, degree of prolapse, sexual life, the desire for additional children and other associated pelvic disorders constitute the chief points on which the decision must be based.

In most instances, surgical repair of the torn or attenuated supportive structures constitutes the procedure of choice. On the other hand, since procidentia occurs more often in elderly women, a considerable number are poor operative risks. For the treatment of this group, some form of mechanical support must be used. Conservative treatment is indicated also in younger women with partial prolapse, who desire to postpone extensive repairs until additional children are borne; similar measures are applicable to the occasional cases of prolapse which occur during pregnancy.

Circular hard rubber pessaries are preferable to the soft or sponge rubber type, as the latter, being rough, readily become encrusted with secretion, are foul in odor, and are more irritating to the vaginal wall. The caliber of the hard rubber pessary should be as small as will maintain the uterus and the associated cystocele in proper position. Ring pessaries of the same caliber rubber as the usual Smith pessary accumulates less encrusted mucus and permit better drainage than those made of larger caliber material. The ring pessary should be large enough in diameter to fill the relaxed vagina and rest comfortably against the pubic rami without making undue pressure on the lateral vaginal walls or the urethra. Occasionally, the vaginal orifice is too small to allow the insertion of a ring large enough to support the uterus; in such cases the collapsible soft rubber pessary must be used despite its disadvantages.

Tampons are useful in maintaining the completely prolapsed structures in position while treating decubitus ulceration of the cervix in preparation for operation. In rare instances, tamponage may prove to be the only method applicable to an elderly patient whose pelvic floor is too relaxed to hold a pessary in place; continued use of tampons, however, has the disadvantage of macerating the atrophic vaginal epithelium.

The cup-pessary, with its attached stem and harness is rarely justifiable. With this device in place, the entire weight of the uterus rests on the cervix, and decubitus ulceration is almost inevitable. Some few patients, who cannot be subjected to operative repair, and who, for various reasons, cannot be fitted with any mechanical support, find considerable relief from the use of a vulvar pad with a tightly applied perineal binder.

SURGICAL TREATMENT

Every gynecologist has his favorite operative methods for correction of uterine prolapse. No one method is suited to all cases, but certain principles are common to all successful operations. The chief cause of prolapse is, unquestionably, over-stretching or tearing of the fibromuscular supportive tissue in the bases of the broad ligaments. Therefore, any procedure which does not include shortening of these structures is doomed to failure. In addition, the almost invariably associated cystocele and rectocele must be repaired, and attention must be given to the frequently associated enterocele.

MANCHESTER OPERATION

This procedure consists of amputation of the cervix, shortening of the bases of the broad ligaments, and anterior and posterior colporrhaphy. The details of the technic of this operation have been so well presented by Fletcher Shaw, of Manchester, and more recently by Frank, Gordon, and Curtis in this country, that there seems no necessity to do so in this presentation. The order in which this combination of procedures has proved most effective and, in our hands, easiest to perform, is as follows: elevation and suture of the bladder high on the anterior face of the uterus, amputation of the cervix, covering the posterior lip of the cervix with Sturmdorf's stitch, cutting away the excess vaginal mucosa, placing but not tying the broad ligament sutures anterior to the cervix, completion of the cervical covering, closure of the anterior colporrhaphy

from the urethral end, and finally tying the sutures previously placed through the bases of the broad ligaments. We have found that tying these chief supportive sutures earlier in the operation often elevates the cervix to such a degree that the remainder of the anterior closure is rendered extremely tedious. Posterior colporrhaphy completes the operation. This is the least radical of all the major surgical measures and is, therefore, best suited to the young woman who desires to have additional children. Shaw found in his follow-up study that labors subsequent to this operation were well tolerated; in only 18.5 per cent was there any recurrence of symptoms. Another advantage is that vaginal depth is more regularly conserved in this procedure than in the more radical measures. The Manchester operation is my choice also for partial prolapse in older women, if there be no special indication for hysterectomy. The shortening of the bases of the broad ligaments may be accomplished with or without detaching them from the sides of the cervix. If the cervical amputation is high, I prefer to detach them and bring them together anterior to the cervix; if the amputation is low, simply overlapping the unsevered ligaments anteriorly serves the same purpose and gives a less "bunchy" anterior cervical lip.

THE INTERPOSITION OPERATION

This procedure was devised in 1898 almost simultaneously by Thomas Watkins[15] and by Wertheim.[16] It consists of longitudinal incision of the anterior vaginal wall in the plane between the bladder and the vesicovaginal layer of the endopelvic fascia, elevation of the prolapsed bladder beyond the line of the peritoneal reflection, incision of the peritoneum of the anterior cul-de-sac, extraction of the fundus of the uterus through this opening, suture of the top of the fundus to the fascia just under the pubic rami, and closure of the anterior vaginal wall so that the base of the bladder rests on the posterior surface of the uterus. Posterior colporrhaphy completes the operation.

Because of the distorted relations of the uterus and the bladder, childbirth after this procedure is hazardous, if, indeed, it is possible. The operation should, therefore, be utilized only after the menopause or artificial sterilization. Its field of greatest usefulness is in elderly women with cystocele and only partial prolapsus. We do not use this procedure in any case in which the cervix protrudes more than one inch beyond the vaginal orifice. The operation in our hands is poorly adapted to the correction of complete prolapse because the relaxed uterosacral ligaments in these cases fail to maintain posterior traction on the cervix. Unless the cervix is held well back in the pelvis, recurrence of the procidentia not infrequently occurs. Care should be exercised that the fundus be sutured firmly enough under the pubic rami to fill the hernial orifice through which the cystocele pushed its way.

The interposition operation is not used in our clinic in young women who are sexually active, because of the high incidence of postoperative dyspareunia.

VAGINAL HYSTERECTOMY

Removal of the uterus is not practiced in our clinic in cases of partial prolapsus unless there be some indication for hysterectomy other than malposition of the uterus. The uterus is the keystone of the upper pelvic arch, and its removal renders shortening of the broad ligaments more difficult. Vaginal hysterectomy is, therefore, rarely utilized electively except in occasional cases of complete prolapse in elderly women.

A brief outline of our procedure is as follows: transverse incision of the vaginal wall just anterior to the cervix; blunt dissection of the bladder from the vaginal wall; longitudinal incision of the anterior vaginal wall from the cervix to a point near the external urinary meatus; elevation of the bladder above the point of peritoneal reflection; completion of circular incision of vaginal mucosa into posterior cul-de-sac; clamping and ligation of both uterosacral ligaments and the lower half of both broad ligaments; severing these ligaments from the uterus; incision of the anterior cul-de-sac, with the

finger in the peritoneal cavity as a guide; delivery of the fundus through the anterior peritoneal opening; ligation of the upper half of both broad ligaments; excision of the uterus; purse string closure of the peritoneal cavity; uniting the severed broad ligaments in the midline; anchoring the united upper ends of the broad ligaments to the fascia under the pubic rami (Mayo technic), just as the uterus is anchored in the interposition operation; attaching the vaginal vault to the united bases of the broad ligaments; anterior and posterior colporrhaphy.

Attention should be directed to the possibility of an associated enterocele, and, if present, the hernial sac must be obliterated or removed. In performing the anterior colporrhaphy on those patients who are incontinent of urine, two or three mattress sutures are taken in the fascia underlying the internal urinary meatus. These sutures should be firm, but should not unduly constrict the urethra; tight constriction often delays the return of normal sphincteric action and prolongs the necessity of catheterization.

PARTIAL COLPOCLEISIS

Partial closure of the vagina by LeFort's operation is the simplest of all the surgical procedures for the correction of complete prolapse. Since, on completion of the operation, the vagina is practically closed, it is limited in its applicability to those patients whose sexual life is of no importance. It is easily performed under local anesthesia; little, if any shock attends the procedure, postoperative complications are rare, and the final results, in the limited number of patients to whom we have found the operation suited, have been eminently satisfactory.

After excluding, by curettage, the possible presence of fundal malignancy, the first step of the operation is a rectangular denudation of the anterior vaginal wall. The denuded area should not be more than 4 centimeters wide, and should extend from a point just below the cervix almost to the urethral orifice. A similar denudation of the posterior wall is followed by a suture of the edges of these identical rectangles, beginning just below the cervix and proceeding toward the introitus. The sutures are placed in pairs, one on each side, so that, as the operation proceeds, the cervix disappears and the prolapsed uterus gradually returns to its normal level. Firm perineorrhaphy is essential for proper support. On completion of the operation, a narrow channel extends upward from each side of the vaginal orifice and these are continuous with the transverse channel just below the cervix. This channel must be of sufficient caliber to provide adequate drainage of cervical and uterine secretions.

The foregoing constitutes our general ideas of the indications for the various operative procedures for the treatment of prolapsus. A brief analysis of 50 consecutive personal cases might be of value in indicating the relative frequency of utilization of the various methods. These, rather than service cases, were chosen because they obviously represent more accurately my own ideas of the subject. The group consists of 24 cases in which the cervix presented at the vaginal orifice and 26 in which a part or all of the uterus protruded beyond the introitus. Lesser degrees of descensus have not been considered in this summary.

The Manchester operation was done in 29 cases and the result was entirely satisfactory in all but two cases in which partial incontinence of urine has persisted.

Vaginal hysterectomy was done in eight cases. In three of these, functional uterine bleeding constituted the indication for removal of the uterus and in one an associated carcinoma of the fundus was similarly treated. The other four hysterectomies were performed electively on elderly patients with complete prolapsus. The results were uniformly satisfactory.

The interposition operation was used in six women in whom the cervix did not protrude beyond the vaginal orifice. The results in this small group have been good.

Partial colpocleisis under local anesthesia, by the LeFort technic, was utilized in seven elderly widows, whose age and general condition contraindicated a longer and more

anatomic surgical procedure. One patient in this group died suddenly while walking in the ward on the seventeenth postoperative day, apparently of cardiac dilatation. The other six patients have been completely relieved.

In closing this brief discussion of the treatment of uterine prolapse, I should like to reiterate that no one method can apply to all cases, and that proper choice of treatment for any patient can be made only after evaluation of many factors.

BIBLIOGRAPHY

1. Hippocrates: Oeuvres completes d'Hippocrate. Traduction Nauvelle par E. Littre. Paris, J. B. Bailliere, 7:317, 1861.
2. Soranus of Ephesus: cited by F. Winckel. A Handbook of Diseases of Women. Parvin's Translation, p. 251. Philadelphia, P. Blakiston, Son & Co., 1889.
3. Rodericus a Castro: cited by Matthew D. Mann. A System of Gynecology, 2:1135. Philadelphia, Lea Brothers & Co., 1888.
4. Pare, Ambroise: Deux Livre de Chirurgie, p. 214. Paris, Chez Andre Wechel, 1573.
5. Blanchard, Stephen: The Physical Dictionary, p. 251. London, Sam Crouch, 1702.
6. Gerardin, Romaine: Archives generales de medicine, 3 me annee Tome 8, 8:132. Paris, 1825.
7. Hall, Marshal: London Med., 9-E. p. 269, 1831.
8. Heming, G. O.: London Med., 17:266, 1835.
9. Geddings: cited by Savage, Lancet, 1:164, 1858.
10. LeFort, Leon; Ann. de Gynec. et d'obst., 7:299, 1877. LeFort, Leon: Bull. gen. de therap., 92:337, 1877.
11. Neugebauer: 2 bl. f. gyn., No. 1 83, No. 2 825, 1881.
12. Donald, A.: J. Obst. & Gyn. Brit. Emp., 28:256, 1921.
13. Fothergill, W. E.: J. Obst. & Gyn. Brit. Emp., 28:251, 1921.
14. Shaw, W. Fletcher: Am. J. Obst. & Gyn., 26:667, 1933.
15. Watkins, T. J.: Am. Gyn. & Obst. J., 15:420, 1899.
16. Wertheim, E.: Zentralbl. f. Gynak., 23:369, 1899.

SIGMOID SINUS THROMBOSIS

REPORT OF A CASE*

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SHREVEPORT

The case presented is one of sigmoid sinus thrombosis which developed as a complication of an acute exacerbation of long standing chronic suppurative otitis media.

REPORT OF CASE

A 22 year old negro man was admitted to Charity Hospital in New Orleans September 1, 1946, complaining of severe shaking chills, high fever and pain in the left ear. He gave a history of chronic, foul, purulent discharge from the left ear for seventeen years. Four days prior to admission to the hospital the otorrhea stopped and the ear became painful. Within 24 hours the patient had a severe shaking chill and high fever. He had two additional chills and exhibited a septic temperature. The otorrhea on the left recurred one day before the patient was admitted to the hospital.

On admission to the hospital the patient was acutely ill. His temperature was 104.4 F. (fig. 1). The copious, purulent discharge filled the left external auditory canal. The ear drum was obscured by granulation tissue polyps. There was neither postauricular swelling nor obliteration of the postauricular fold. However, exquisite tenderness was found over the mastoid antrum and this extended posteriorly over the sigmoid sinus, suggestive of a perisinus abscess. Palpation revealed deep tenderness in the neck along the left internal jugular vein. There was moderate nuchal rigidity but no other evidence of meningeal irritation. Neither nystagmus nor past pointing could be found. Weber's test showed lateralization to the left ear; the result of the Rinne test on the left was negative.

Examination of the blood revealed 15,000 white blood cells per cubic mm. of blood with 87 per cent polymorphonuclear leukocytes. No malarial parasites were found. Blood cultures gave negative results on September 5 and 6. Examination of the spinal fluid on admission disclosed no increase in pressure, clear fluid and no cells. The sugar was 74 mg. per cent and chlorides 655.2. The Tobey Ayer test was not done. The only other abnormal finding was a soft blowing systolic precordial murmur. The electrocardiogram was normal.

A tentative diagnosis of sigmoid sinus thrombosis was made. Before operation was performed the infection was combatted by the administration of 20,000 units of penicillin every three hours and, after a large initial dose, 1 gram of sulfadiazine every four hours. However, six days of this therapy did not alter the course of the disease.

On September 6, endaural radical mastoidectomy was performed (fig. 1). A large perisinus abscess and an abscess within the sigmoid sinus were found. The distal and proximal ends of the sigmoid sinus were thrombosed. Contrary to the standard accepted methods of treatment only the sinus and perisinus abscesses were drained and the thrombus of the distal and proximal ends was not disturbed.

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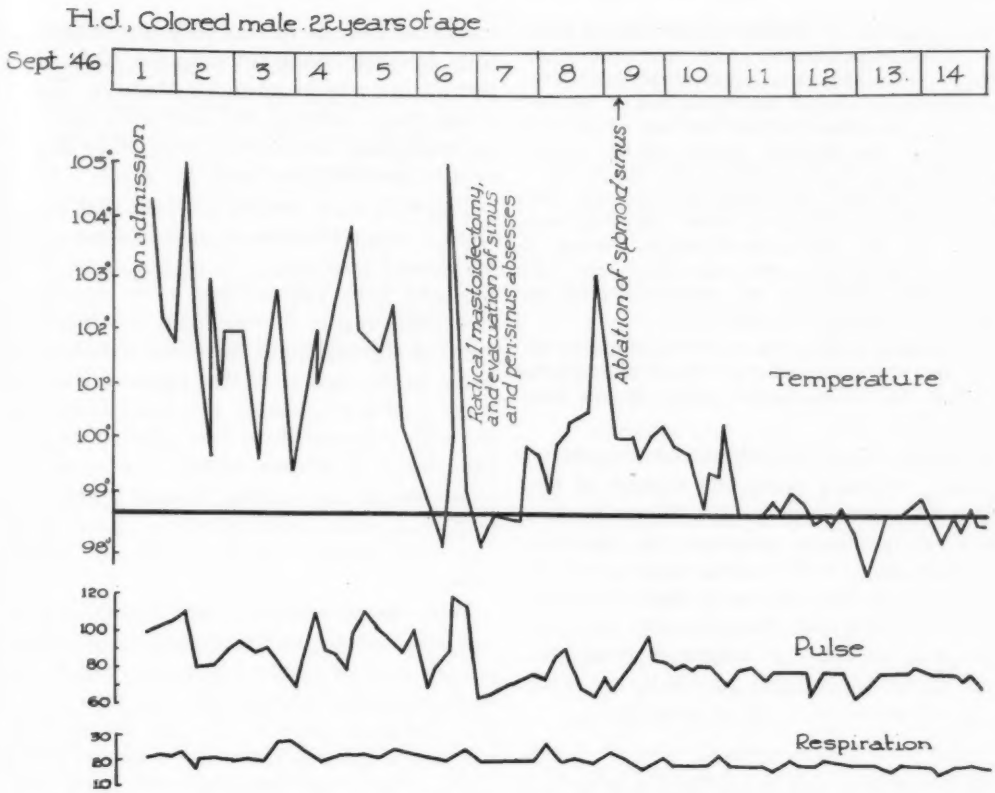

Fig. 1 is the graphic temperature chart of this case of sigmoid sinus thrombosis caused by a gram-negative bacillus. There was no response to five days of intensive sulfadiazine and penicillin therapy. Surgical drainage on the sixth hospital day produced temporary remission. However, the temperature did not subside until the septic thrombus was removed from the sinus.

Culture of the abscess revealed a gram-negative rod, which was not viable on sub-culture and could not be identified. In all probability it was *Bacillus proteus*. In any case, it was clinically resistant to chemotherapy.

The temperature remained normal for nearly 24 hours following evacuation of the abscessed cavity. Within 48 hours postoperatively the patient's temperature was 100.4 F. and before the patient reached the operating room the next morning, it had risen to 103 F.

On September 9, 1946, the mastoid was reopened through a classical postauricular incision with an accessory perpendicular incision extending backward over the transverse sinus (fig. 1). Through this incision the perisinus abscess was widely exposed until healthy dura was obtained on the medial and posterior aspects of the perisinus abscess. To obtain bleeding from the upper end of the sigmoid sinus it was necessary to expose and open the anterior third of the transverse sinus and evacuate the septic thrombus with a catheter placed on the end of the suction tube. Free and copious bleeding was obtained from the distal end of the sinus and the sinus was allowed to bleed sufficiently to wash away all septic clots. No bleeding was obtained from the proximal end of the sinus although the catheter was inserted through the jugular bulb into the upper end of the jugular vein. The wound was packed with iodoform gauze and the postauricular incisions were closed. Twenty-four hours later, at the end of the tenth hospital day the patient's temperature reached normal and remained thus for the month during which the patient was in the hospital for dressings and postoperative care.

The patient continued to receive penicillin and sulfadiazine for five days after the temperature became normal—a total of sixteen continuous days of sulfadiazine and penicillin therapy. During this period the average daily fluid intake was charted as 4,000 c.c., the average daily urine output was charted as 3,250 c.c. In the presence of the gram-negative rod it is questionable whether chemotherapy in any way altered the course of this infection.

From the time of ablation of the sigmoid sinus, the patient's postoperative course was entirely uneventful. Five days after ablation the packs were changed under general anesthesia and at regular intervals thereafter without the use of general anesthesia. To facilitate healing of the radical mastoid cavity a split-thickness skin graft, taken from the left leg was placed over the roof and medial walls on October 3, 1946. No graft was placed over the still infected region. Where it was applied, the skin graft took 100 per cent. At the patient's request he was discharged from the hospital on October 10, 1946.

He returned to his home where his physician reports that his convalescence continued uneventfully and that the radical mastoid cavity is now completely dry.

Sigmoid sinus thrombosis has become a medical curiosity since the advent of the sulfonamides and penicillin. When it does occur, it no longer presents the classical symptomatolgy and findings and is not accompanied by the previously high mortality rate. With adequate chemotherapy the otologist may in time be taught to treat sigmoid sinus thrombosis medically or with more conservative surgical procedures.

DISCUSSION

Sulfonamides and penicillin are effective in two different ways. First, they control infections of the middle ear and mastoid, consequently greatly reducing the incidence of mastoiditis and its complications. Secondly, these drugs have an inhibiting effect on the surgeon. Mastoid infections are no longer regarded as emergencies. The eager surgeon no longer operates immediately on acute mastoiditis, thus spreading the infection in all directions. These drugs have given the otologist equanimity.

Chills, spiking temperature, high white blood count and a positive blood culture in the presence of an acute or chronic infection of the ear were formerly the characteristic findings in sigmoid sinus thrombosis. Rarely does one now have the opportunity to take a blood culture before the patient has received some drug which makes the blood culture negative and the white cell count normal. These drugs often completely mask the infection so that classical findings of sigmoid sinus thrombosis do not occur until the therapeutic measures have been discontinued; or, as in this case, the infection may be drug resistant and the fever will persist in spite of adequate therapy. These drugs have reduced the mortality rate of sigmoid sinus thrombosis from 30 to 40 per cent to around 5 per cent.

It may arouse debate to state that early sigmoid sinus thrombosis may in some cases be treated medically. All of you have seen patients with fulminating acute suppurative otitis media in whom all the classical signs of sigmoid sinus thrombosis developed early in the course of the acute infection. Such patients probably present periphlebitis with a mural thrombus sending emboli into the blood stream. In the absence of destructive or necrotizing disease I am certain that many such infections have been and will continue to be cured by drugs alone.

With the chemotherapeutic measures acting as a barrier to the spread of infection it may be that in the future we will learn that established surgical rules may be modified and that sigmoid sinus thrombosis will respond to more conservative measures. Recently, a personal communication from Verling Hart of North Carolina told of a case of sigmoid sinus thrombosis which he cured by simply evacuating the abscess within the sigmoid sinus, no effort having been made to obtain free bleeding from the distal or proximal end of the sinus. In the case reported here this more conservative measure was courageously tried at the first operation without avail. The infection did not respond until the sigmoid and transverse sinuses were opened and free bleeding was obtained from the transverse sinus. It was, however, unnecessary to ligate the internal jugular vein. But, jugular ligation is of proved value when the infection has not responded to simple ablation of the sigmoid sinus. Nevertheless, there will always be cases of sigmoid sinus thrombosis which must be treated surgically because of drug resistant organisms and the necessity for drainage of abscess cavities.

Gerzog[1] and Dearmin and Sims[2] have reported cases of thrombosis of a lateral sinus caused by the *Bacillus proteus*. They make

the following points relative to the otogenic behavior of *Bacillus proteus*. First, the site of infection is usually a chronic discharging ear, the bacillus acting as a secondary invader in most instances. Secondly, the vascularity of the field appears extremely inviting to the organism with the result that perisinus abscesses and lateral sinus thromboses are frequent. Finally, the progression in these cases is woefully extensive, meningitis and abscess of the brain often resulting.

CONCLUSIONS

When sigmoid sinus thrombosis is suspected, we have adopted the following arbitrary rules: (1) An attempt is made to obtain blood cultures and blood counts before instituting any chemotherapeutic measure. (2) The infection is given an adequate trial on a course of combined sulfonamide and penicillin therapy with all supportive measures (if possible at least three days). (3) If sigmoid sinus operation is indicated, any perisinus abscess should be widely exposed until normal dura has been obtained in all directions. The sigmoid sinus is then opened and an attempt is made to obtain free bleeding from both extremities if necessary, going posteriorly to the torcula to obtain free bleeding from the transverse sinus. (4) Ligation of the internal jugular is reserved for those cases which do not respond to ablation of the sigmoid sinus. (5) The patient is given adequate sulfonamide and penicillin therapy until the temperature is normal for at least five days.

REFERENCES

1. Gerzog, B. G.: Bacillus proteus and otogenic infection; secondary mastoiditis, thrombosis of the lateral sinus and bacteriemia, Arch. Otolaryng., 30:275, 1939.

2. Dearmin, R. M., and Sims, J. L.: Thrombosis of the lateral sinus and abscess of the temporal lobe due to bacillus proteus, Arch. Otolaryng., 32:1098, 1940.

FACIAL NERVE PARALYSIS RESULTING FROM FRACTURE OF THE TEMPORAL BONE*

REPORT OF A CASE

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Facial paralysis, the most grotesque complication of disease of the temporal bone, most often results form a fracture of the temporal bone, Bell's palsy, acute or chronic otitis media and mastoiditis or operative injury. Ballance and Duel[1] have stated that the time to operate upon facial paralysis is now, "now" being the earliest possible time that the patient can be prepared for operation. This opinion is subject to debate among otologists. The following case is presented to introduce the subject of facial paralysis and to clarify the indications for surgical intervention.

CASE REPORT

R. B., a 22 year old negro man, was admitted to Charity Hospital in New Orleans on August 9, 1946, complaining of a profuse, purulent discharge from the left ear and facial paralysis on the left side.

On June 15, 1946, the patient received a severe blow behind the left ear. He was brought to Charity Hospital unconscious, there was bleeding from the left ear and the left side of his face was completely paralyzed. He remained unconscious for six days. Lumbar puncture revealed grossly bloody spinal fluid. The Kahn reaction was positive. To prevent infection the patient was given 800,000 units of penicillin. He recovered from the skull fracture and when he was permitted to leave the hospital, his only complaints were a discharging left ear and facial paralysis on the left.

Otologic examination on readmission August 9, 1946 revealed a copious purulent discharge which entered the left external auditory canal from a fistulous tract through the posterior superior bony canal wall. A probe could be inserted through this

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fistula to the region of the mastoid antrum. The fistula was surrounded by spicules of dead bone. The drum membrane was intact and relatively normal. The patient had fair hearing in the left ear.

All the muscles of the left side of the face were completely paralyzed. Special tests revealed loss of taste on the anterior two-thirds of the left side of the tongue. Faradic stimulation of the trunk of the facial nerve produced no response. There was slight response of the obicularis oculi and quadratus labii inferioris to faradic stimulation. The response was sluggish to galvanic stimulation of all branches.

Roentgenograms revealed diffuse clouding of the left mastoid and evidence of the old fracture into the temporal bone. The blood Kahn reaction remained positive in spite of the previous administration of 800,000 units of penicillin.

On August 15, 1946, left simple mastoidectomy was performed and the vertical course of the facial nerve was decompressed. All the mastoid air cells were filled with purulent exudate. There was moderate bone destruction and excessive bleeding throughout the procedure. The fracture of the temporal bone had extended through the roof of the antrum and had involved the dura. As a result, the temporal lobe herniated into the mastoid antrum and the superior portion of the mastoid cavity. The herniation measured 1 by 1 by 2 cm. The brain hernia was retracted to complete the mastoid operation. Exploration of the infralabyrinthine area revealed a comminuted fracture immediately below the posterior semicircular canal. Decompression of the vertical course of the facial nerve disclosed several small spicules of bone impinging upon the medial and posterior surfaces of the facial nerve immediately below the horizontal semicircular canal. These spicules were removed. The underlying portion of the facial nerve was contused and edematous.

The small fistulous tract into the external auditory canal was debrided and the bony sequestra were removed. One hundred thousand units of penicillin was placed in the mastoid cavity and the upper two-thirds of the postauricular incision was closed. A Penrose drain was placed in the wound extending down to the region of the mastoid antrum. Postoperatively, meningitis developed. This responded to the administration of 3,500,000 units of penicillin intramuscularly and 160,000 units intrathecally.

At the time of this report, five months postoperatively, the patient has serviceable hearing in the dry left ear and there is partial return of function to all the muscles of the left side of the face (fig. 1). Complete recovery of this facial paralysis is anticipated.

DISCUSSION

We believe that decompressing the facial nerve and removing the impinging spicules of bone facilitated recovery of function of the facial nerve in this case. The indications for the simple mastoidectomy are definite; one may debate the indication for decompressing the facial nerve. Possibly, the function would have been recovered at the same speed without surgical intervention. However, to wait for spontaneous recovery would mean to wait for six months to a year for return of function without knowing the condition of the nerve which may have been either contused or partially or completely lacerated. Decompression is believed to benefit a contused nerve. In the case of a cut nerve procrastination is futile. Therefore, early exploration of any facial paralysis resulting from fracture of a temporal bone is the treatment of choice.

Surgical treatment for Bell's palsy has been subject to considerable debate. Simple Bell's palsy is believed to be of two types, peripheral and central. The peripheral type is thought to be caused by edema of the facial nerve at the stylomastoid foramen. The central or herpetic type (not to be confused with central facial paralysis) involves the geniculate ganglion and has associated disturbances of lacrimation and sweating. Surgical treatment is not advocated for the central type of Bell's palsy. Some authors recommend early decompression of the facial nerve at the stylomastoid foramen in the peripheral type of Bell's palsy. We have seen few cases of Bell's palsy which did not progress to spontaneous recovery. We, therefore, do not believe surgical treatment is advisable in these cases unless the paralysis fails to recover or unless there is loss of response to faradic stimulation at the end of three months' observation. Such cases are rare.

Facial paralysis developing in acute suppurative otitis media is probably secondary to localized neuritis resulting from spread of infection through a natural dehiscence in the fallopian canal. Such facial paralyses which develop in the first two weeks of an acute attack of otitis media do not warrant

surgical intervention since they respond to the treatment for otitis media.

Facial paralysis which develops during the latter or destructive phase of acute suppurative otitis media warrants immediate surgical intervention. In acute suppurative mastoiditis simple mastoidectomy will usually suffice. In chronic suppurative mastoiditis radical mastoidectomy should be performed with removal of all the pathologic process surrounding the exposed facial nerve.

Postoperative facial paralysis appearing immediately after the patient recovers from the anesthetic should be explored promptly. Facial paralysis which develops a few days postoperatively will probably recover spontaneously although some surgeons advise decompression.

Operation on the facial nerve may consist of a simple decompression of the nerve, of decompression and end-to-end anastomosis of a cut nerve or of nerve grafting. Decompression of the facial nerve at the stylomastoid foramen is advocated for the peripheral type of Bell's palsy. Decompression of the injured portion of the facial nerve is advisable in temporal bone fractures. Decompression and end-to-end anastomosis of a facial nerve may be performed if only 1 to 2 mm. of the nerve substance has been lost. Decompression and rerouting of the facial nerve in a new fallopian canal cut along the anterior wall of the external auditory canal is recommended if up to 23 mm. of nerve substance has been lost. Most surgeons, however, prefer to replace this lost nerve by a nerve graft into the fallopian canal.

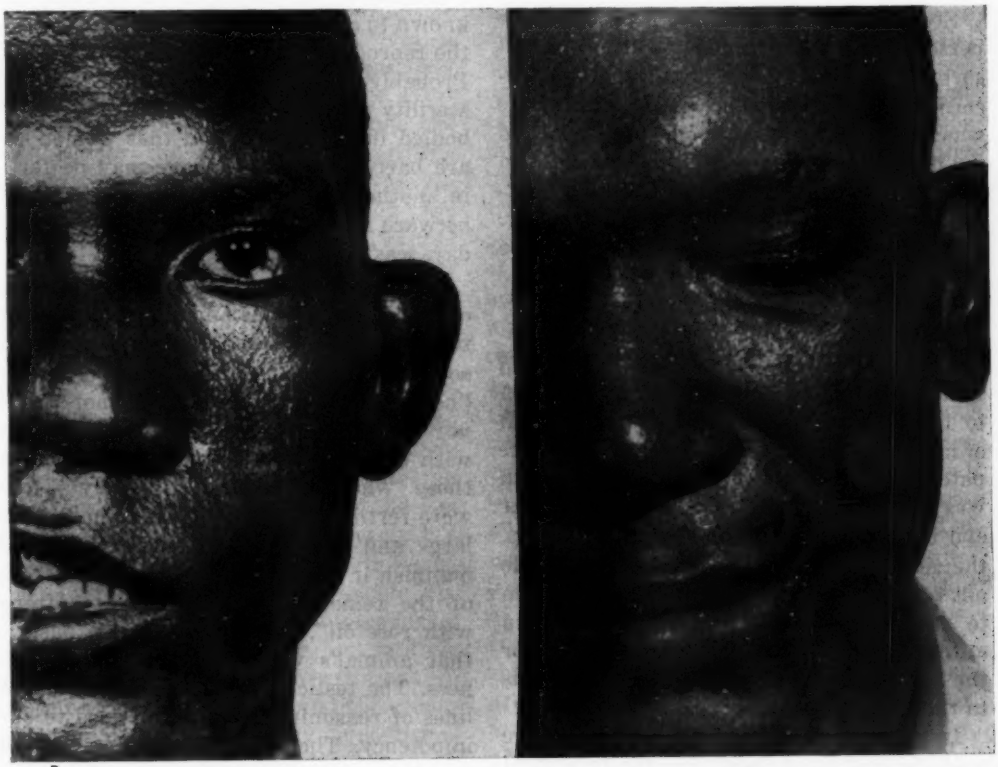

Fig. 1. The photograph on left (A) demonstrates left complete paralysis of all branches of facial nerve. The photograph on the right (B) demonstrates the return of function of all branches of facial nerve following decompression of the vertical course of the nerve and the removal of impinging bone spicules.

COMMENT

Regardless of the method used intratemporal restoration of the continuity of the facial nerve is to be preferred to anastomosis with the glossopharyngeal or the spinal accessory nerve, as the plastic surgeon's correction of the deformity, for intratemporal operation is the only hope of restoring bilaterally coordinated emotional expression.

REFERENCE

1. Balance, C., and Duel, A. B.: Operative treatment of facial palsy by introduction of nerve grafts and fallopian canal and by other intratemporal methods, Arch. Otol., 15:1, 1932.

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A SURVEY OF GENERAL AND OVARIAN STERILITY WITH SPECIAL REFERENCE TO ANOVULATION*

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Sterility has been a problem of grave personal and social import, and one of absorbing medical interest, throughout recorded history. The plight of the sterile wife was always an unhappy one, for, tortured by her longing for a child, she was also doomed to be ostracized, abandoned, beaten, even burned alive by primitive society. Infertility was held to be a peculiar crime of females, and the reasons behind the harsh punishment meted out to the unproductive one were of a magical and an economic nature. Offspring represented wealth, for every child meant an addition to the available labor, and a bigger farm or a greater herd thereby accrued to the paterfamilias. Moreover a sterile woman was bewitched, and the contagion of her evil barrenness caused the crops to dry up, the orchards not to bear, and the cattle not to multiply, but to sicken and die. Even to recent years the Baganda of Africa authorized divorce of a childless wife for the reason that her sickness is contagious to the fruit trees and crops.

In later times, when some attempts were made to find the reasons and the treatment for sterility, the wife had to undergo diagnostic and therapeutic ordeals scarcely less formidable than the alternatives of the savage days. The history of the ideas on origin and therapy of sterility, as outlined in Ricci's *Genealogy of Gynecology*, is fascinating to follow:

Hippocrates (460-377 B. C.) listed as causes for sterility, malpositions of the cervix, excessive smoothness of the lining of the uterus, prolapse, obstruction of the os ("the veins of the uterus becoming so large they do not retain the semen") and profuse menstruation ("the blood vessels are too weak to hold the semen").

Physicians of the Alexandrian school founded around 307 B. C. were indebted to Aristotle for many of the still valid observations he made on the physiology of fertility. He states that menstruation usually begins at 13, ceases at 40, but had been known to continue up to age 50; he bounded the reproductive epoch by these two events. Probably the first recorded idea that male sterility is worthy of consideration is embodied in his observation that women who are barren in one marriage may be fertile in another. The question of incompatibility between sperm and ovum, undecided to this day, was raised at that time.

Around 2 A. D., in the medical writings of Rome were appearing the beginnings of the recognition of endocrine syndromes associated with barrenness. If women were "not too long nor too thickset, not too fat nor too flabby, not too moist nor too dry, with cervices not too open nor too closed, those who menstruate regularly"—these were fertile. Also "those who do not possess large and fleshy loins, and those who are mannish in their physique are sterile." One of the remedies was "lion's fat softened with rose oil", on the presumable basis of that animal's vaunted potency and maleness. The testicles of an ass, along similar lines of reasoning, were being used to cure impotency. These crude harbingers of organotherapy were but a sample of that vast and inconceivably ancient body of lore known to anthropologists as homeopathic magic—the procedure of attempting to

*Awarded third place in the Schering Corporation's 1946 national essay contest on "The Role of Hormones in Sterility."

bring about a phenomenon by the imitation thereof, usually assisted by powerful incantations. The same principles are applied by quacks today, and curiously enough, when intelligently applied by some of our reputable hormone theorists, are achieving some success today in the form of cyclic endocrine therapy.

Next to occupy the historical scene is Abn Ali Husayn Ibn Sina Avicenna (980-1037), the giant of Arabian medicine, and indeed, of European for five centuries. An enormous dissipator and equally prodigious writer, he turned out twenty major treatises. Chief among these is the famous *Canon*, in which appear the luminous words destined to Cassandra's fate for the next thousand years: "Sterility is due to causes involving both the male and the female." Psychic disorders were also invoked by this most discerning of savants, and diseases of the "sperm" of both partners. Some of his less illuminating comments reveal a bizarre misconception of female anatomy probably rivalled only by that of the average modern businessman, for example the procedure for the diagnosis of sterility: "The older method of fumigating the vagina with aromatics and directing the fumes into the matrix by means of a funnel (traitectario) is preferable; if the aromatic odor does not exhale from the mouth or nose there is sufficient indication that the woman is sterile."

Passing lightly over a large and much muddled and misguided era characterized by mass malpractice and religious suppression of all original publication differing seriously with Galen, we arrive at the Renaissance. Here the pendulum reached a new amplitude of magical speculation and fancy, and the original, often keenly observant methods of the ancients were largely submerged in the highly imaginative compounding of esoteric prescriptions to awe the unlettered and confound the credulous. Some of the more intelligble nostrums included rabbit saliva or mare's milk drunk immediately before coitus; Johannes de Ketham (c. 1460) considered tampons soaked in oil with the powdered right testicle of a weasel to be remarkably efficacious. Sennert of Wittenburg (1572-1637), a leader of the psychosomatic trend of that day, observed that sterility was sometimes due to "imbecility of the uterus."

Around 1709 Carlo Musitano prescribed obscene entertainment for sterility, and as late as 1739 the view was publicized that if a woman stood in the right place, at the opportune time where animalculae were floating in the air, and the west wind was blowing, she became pregnant.

All was not darkness, however, in the eighteenth century's concepts of sterility. Perhaps the new dawn of the logical approach to this problem was seen in a treatise by Martin Naboth, written in 1707, and mentioning as etiologic conditions sclerotic ovaries and closed tubes. This work was written around the beginnings of the anatomic-histologic approaches to all medical questions. Morgagni gave a valuable resumé of the anatomic factors in sterility, including sclerotic ovary, absence or diseases of ovarian follicles, anomalies of vagina and external genitals, absence of uterus, obliquities of uterus, and occlusion of the tubes.

Parallelling the halting advance in the gynecologic study of infertility, the study of sex hormone physiology was highlighted by the idea of Thomas Willis (1622-1675), who suggested that puberty occurred as a result of ferments which generated in the gonads and passed into the blood stream. Bichat furnished experimental proof that the secretions of the endocrine organs were esssential to vital activities by his observations of the effects of testicular and ovarian secretions on spayed animals and eunuchs. The same year, 1799, John Hunter performed the first artificial insemination of a woman. DeGraff's studies on the ovarian follicle and corpus luteum and Cruikshank's investigation of ovarian tubal migration placed reproductive physiology on a firm foundation, upon which rests much of the experimental work that has followed.

Hormonology progressed apace, with the description by Berthod of the psychosomatic effects of castration, (1849), and the

experiments of Brown-Sequard with testicular extract at the turn of the century.

During the first ten years or so of this century hormone studies consisted of experimental, largely histologic, work on animals. Following this were several years of research with crude glandular extracts, after which the biochemists began to describe the chemical nature of these products.

The first evidence of ovarian hormone action other than luteal was discovered by R. T. Frank in 1917, when he observed the effect of follicular fluid on the endometrium.

Smith and Engle, and Ascheim and Zondek, working independently, published in 1927 the first description of the gonadotropic function of the anterior pituitary. The gonadotropic activity of the serum of pregnant mares was brought to light in 1930 by Cole and Hart, and a year later the separation of anterior pituitary hormone into "Follicle Stimulating Hormone" (F. S. H.), and "Luteinizing Hormone" (L. H.) was announced by Fevold, Hisaw, and coworkers. Wassen-Lawrence published studies on F. S. H. and L. H. not long afterward. Moore and Price confirmed the findings of Smith and Engle in 1932. Demonstrating their findings by laparotomy, Davis and Koff laid the foundation of gonadotropic therapy in 1937 when they showed that ovulation in women could be induced by the intravenous administration of "Pregnant Mare's Serum" (P. M. S.).

Turning again to the subject of sterility investigation, the roster of contributors in the twentieth century is studded with famous names. To Huhner goes the credit for the post-coital test of seminal adequacy that turned the spotlight of diagnostic inquiry full upon the husband for the first time. G. L. Moench taught us most of what is known about abnormal morphology and activity of spermatozoa, while Cary and Rubin contributed tubal insufflation for the diagnosis of the biggest factor in female sterility.

Burr, Rock, Hill, Allen, Rubinstein, Zuck, and their co-workers are credited with several excellent methods of timing the day of ovulation. Hartman's experimental work on ovulation and the clinical studies of Ogino and Knaus on fertile and sterile periods in relation to ovulation are classics. Novak's description of the anovulatory cycle, and Hamblen's interpretations of endometrial biopsy for its diagnosis and his cyclic gonadotropic and steroid therapy for its correction, have well begun the solution of an as yet tangled problem. Clinical studies have been remarkably facilitated by Dickinson's contributions in sexual anatomy and Meaker's crystallization of what is known of a complex subject in his masterfully conceived diagnostic schedules. To mention with praise all the outstanding workers in the field of sterility needs a tome, and to do justice to what has been accomplished in this century alone, several volumes—yet the developments in the endocrine aspects of the subjects are in embryo, and vast possibilities are still untapped for research in prevention.

The barren marriage is still a personal and social problem of pressing concern. Magnitude of the public aspects of it would appear to be increasing: There is a 200 year old downward slope in birth rate in this country, interrupted only by the spurts of increase during war times, and a steep decline over the last fifty years. Decrease in birth rate in itself constitutes no threat of depopulation in the United States, but there has been found to exist a disadvantageous scatter of fertility among the various population groups. It is clear that those least fitted by education, health, and economic advantage to have offspring have many more than those ideally prepared for reproduction. Group and racial differences in fecundity occur along geographical, social, religious, and economic lines, and most are explicable on the basis of the ability to obtain, and knowledge of, the best contraceptive measures. There is no observed hereditary fertility differential.

The reasons for the relative infertility of the upper bracket are ill health and voluntary restriction in order to meet real or artificial social and economic circumstances, the latter motive being the greatest

single factor. The professional, business, and skilled labor groups in particular marry later and delay having offspring longer than do the other groups, who seem to disregard illness as a contraindication to pregnancy, and who by reason of lack of contraception, education, or other diversion, tend to exploit their prolific propensity. Voluntary sterility, however, is germane to this discussion only in that it not infrequently becomes involuntary when prevenception is practiced past the peak fertility years of the late marrying couple.

All factors considered, around 17 per cent of marriages in the United States were childless over the period 1910-1930 and around 12 per cent of all marriages are involuntarily barren.

The fact that the terms "barren marriage" and "sterile mating" are so frequently used instead of "male sterility" and "female sterility" is a credit to the men who initiated the bilateral sterility studies, and especially to Meaker, who so clearly set forth the principles of the etiology of sterility: First, in the vast majority of cases of human infertility the cause is not a single one, but the product of several factors. Depending upon the relative importance assigned by different authors to the various factors causing infertility, there are 2.23 to 4.79 abnormalities per couple.

Next, the several conditions diminishing fertility in each case are very rarely limited to one partner: only 10 per cent of the husbands and 5 per cent of the wives investigated showed no evidence of decreased fertility. Defects imposing absolute sterility were found in only 30 per cent of cases, while 70 per cent showed no single causative condition. When present, defects such as azoospermia or closed tubes, imposing absolute sterility are divided about equally between the two partners. Where the inability to reproduce is relative, the male is at fault (chiefly) in 25 to 50 per cent of barren matings. Of all demonstrable abnormalties detrimental to fertility about 70 per cent are on the distaff side, which is not to say that the wife is responsible in 70 per cent of cases.

Finally, the multiple sterility factors consist of genital disorders and those of a constitutional nature. A slight diminution of fertility from constitutional causes in one partner may so multiply a similar fault in the other that the union is unproductive, although either party is still fertile in the sense that offspring might be produced in a mating with another partner of high fertility.

Much ink and fine foolscap have been lavished upon the proper definition of sterility, but for all practical purposes it is the inability to produce living chilren after honest effort has been made for several years. Strictly speaking, female sterility is the inability to conceive after two or more years of marital intercourse, in the absence of prevenception, with a normal man. Since the reproductive function of the ovary extends past the moment of conception, ovarian sterility is less simple to define; it may be designated as failure of conception, or of early nidatory maintenance of the fertilized ovum, by reason of deficiency or failure of the several ovarian functions.

The foundation of diagnostic methods and the rationale behind therapies directed against sterility consist of the laboriously evolved and now widely accepted ideas of generative development and reproductive physiology. It is assumed that the reader has already been exposed to all the accepted as well as the unaccepted notions on the subject—so we shall not burden him further therewith. Suffice it to say that the reader should bear constantly in mind all the steps in the developmental, anatomical, and functional history of the male and female reproductive systems, as well as all the facts he can muster on hormone physiology of same—throughout the rest of this discursion.

Throughout its development and the fulfillment of its destiny the reproductive system more than any other is constantly influenced by its own internal secretions, those of the other endocrine glands, and the fluctuations in the level of health and general metabolism. Disorders of the endocrine glands are partly or entirely at

fault in 20 to 57 per cent of cases of sterility. What the incidence is of sterility from constitutional depression is not known, but there are some figures available on the incidences of flaws arising at the many other vulnerable links in the complex chain of reproduction:

I. Male factors: It has been stated that 90 per cent of husbands of sterile matings have some impairment of fertility, and that the male is chiefly at fault in 25 to 50 per cent of such cases; there remains to break down these estimates into the separate defects themselves.

A. Testicular function: 80 per cent show some seminal impairment, severe enough in 20 per cent to impose absolute sterility.

B. Occlusion of ducts: 5 per cent of males of sterile matings.

C. Factors of unknown incidence are those of hostile secretions, ejaculative, and other coital elements. Constitutional depressions; for example, prolonged wasting diseases, seem to lower male fertility more than any other factor. H. W. Rowe cites the following incidences of contributing endocrine causes in 100 males chosen from sterile matings:

Pituitary disorder	35
Thyroid	21
Unclassified	2

II. Female Factors:

A. Tubal abnormalities: The largest group of causes. Incidences range from 43 to 64 per cent among sterile women; there may be absence, hypoplasia, or obstruction, the latter being most frequent.

B. Uterine abnormalities: Obstructive and infectious lesions 2 per cent; endometrial tuberculosis 5 per cent; fibroids 12 per cent.

C. Cervical factors: Cervicitis 22 to 49 per cent; poor drainage 4 to 6 per cent; malposition of cervix 4 per cent.

D. Endocrine factors other than ovarian: In 100 women of barren matings H. W. Rowe found pituitary dysfunction to be contributory in 34, and unclassified endocrine disorder in 2 per cent. It is certain that the incidence of endocrine causes is great, but most of their effects are mediated through the pituitary or ovary.

E. Factors of unknown incidence: Coital difficulties—Here the presenting complaint is not usually that of sterility. Abdominal factors acting to prevent the ovum from entering the tube. Constitutional, infectious, metabolic, endocrine, toxic, and psychic disorders directly or indirectly depressing ovarian function in any way.

F. Ovarian factors: It is possible for the ovary to fail in any or all of four functions. These constitute the estrogenic, the gametogenic, the ovulatory, and the progestational or luteal. From 20 to 25 per cent of all female sterility is of a functional nature; beyond this little is known of the incidence of ovarian sterility with the exception of that type resulting from failure of ovulation. Opinions differ widely on these figures; it is certain that not all of the authors use the same criteria or even the same definitions of this condition; the true incidence is probably higher than reports from sterility studies would indicate, since many women come in with the complaint of irregular, prolonged, or excessive bleeding when they are afflicted with anovulatory sterility as well.

The percentage incidence of the condition varies from 13 to 54 per cent, but the consensus runs at about 15 to 35 per cent.

About 30 per cent of sterile women presenting no other abnormality have cyclic anovular menstruation, whereas only 2 per cent of normal women are thought to show this dysfunction. In the presence of regular menses up to age 40 the chances of any one cycle being anovulatory are less than one in ten. Even in the ages 40-49, about 50 per cent of the cycles are ovulatory. No significant decrease in fertility should be effected by anovulatory cycles unless they outnumber the ovulatory.

Sterility arising from ovarian failure outranks all other types in challenge and intrinsic interest, as well as in the high proportion of hypothesis inherent in most explanations of its etiologies. It is common knowledge that sterility is often associated

with severe endocrine disease, as the pituitary or adrenocortical cachexias, giantism, acromegaly, adiposogenitalism and marked hypothyroidism; and even in these conditions the real reasons behind sterility are mostly unknown. Our concern is not with the endocrine disasters, however, for few such cases appear with the primary complaint of sterility, and if they should do so many would be judged unfit for parenthood and no therapy directed against their barrenness. Most women afflicted with endocrine sterility have one of several disorders of glandular function, usually not of an obvious nature, and the underlying factors operating against the ovary in these cases are often quite obscure. With this division of the question, then, where speculation is rife, lies our interest; in its study the following general principles have gained some acceptance:

1. Proper ovarian function is dependent upon gonadotropic stimulation by the anterior pituitary and the ovary's ability to respond.

2. When response is absent or deficient to the point of sterility the condition is called primary ovarian failure, prefixed with "early" or "late" depending upon whether mature response failed to eventuate or did so and was later lost. This concept of ovarian refractiveness is based upon the very definition of a hormone as a "chemical substance produced by glandular activity, liberated into the blood stream, and transported to distant parts of the body where it is capable of exerting catalytic action, the degree of which is determined by local electrochemical conditions existing in the cells at that particular time."

3. When stimulation by the anterior pituitary is absent or deficient to the point of sterility, the condition is known as secondary ovarian failure.

4. The chief direct cause of sterility in both primary and secondary failure is absence of ovulation. In view of that fact, the major portion of the remainder of this discussion is directed toward the causes, the diagnosis, and the treatment of anovulation, as it occurs in primary and secondary sterility. The latter may be thought of as anovulatory sterility chiefly due to loss of function of the anterior pituitary, then, while primary ovarian failure is sterility of mostly anovular character, due to inability of the ovary to respond to the gonadotropins.

In both primary and secondary ovarian sterility, the failure may occur in any or all of the four previously mentioned activities: the gametogenic, the ovulatory, the estrogenic, and the luteal, or progestational. It is clear that estrogenic failure necessarily implies failure in the rest of ovarian activity, and that luteal deficiency is included likewise in ovulatory failure. Sterility from purely gametopathic or luteal causes is possible and does occur to a lesser extent, while either type may be caused by most of the disorders giving rise to general ovarian failure. Actually, with the exception of some local changes adversely affecting the ovary or pituitary gland, the postulated causes of all types of ovarian sterility are similar to or identical with the causes of anovulation; so they are best discussed together to avoid undue repetition. The safety of this assertion is almost guaranteed by the paucity of definite knowledge as to the exact mechanisms by which these etiologic agents operate, and the true importance and incidence of each. These etiologies fall into endocrine and extra-endocrine categories, some affecting the ovary, the pituitary, or both, either by directly effecting local anatomic or functional damage or by constitutional depression, indirectly.

A rapid review of the endocrine glands not directly associated with the reproductive apparatus will perhaps illuminate the logic behind some of the charges levelled against these organs as contributors to ovarian failure.

1. *Thyroid*: The secretory activity of the thyroid gland is largely governed by the anterior pituitary's thyrotropic secretion (which, incidentally, is believed to be identical with L.H.), and possibly by neural stimuli from the peripheral and special sense organs and the cerebral cortex, all acting through the hypothalamicopituitary

pathway. Instances supporting the latter view are the enlargement at defloration, and the onset of exophthalmic goiter shortly after an emotional crisis. Hyperthyroidism is only rarely mentioned in sterility studies; hypothyroidism, however, is regarded as a large contributor to the disorder in several ways. The systemic depression can directly alter ovarian metabolism or indirectly alter it through its effect on the pituitary. The actions of the thyroid, the deficiencies of which are most often invoked to explain adolescent ovarian failure, anovulation, luteal deficiency, and gametopathy are the calorigenic (chiefly), then the protein mobilizing action, then the glycogenolytic. Gametopathic fault is assumed, but never proved, in cases of low-grade hypothyroidism where sterility or repeated abortions occur.

2. *Adrenals:* It is thought that disordered adrenal function may bring about ovarian sterility in the following ways:

a. The constitutional impairment of water and electrolyte metabolism may depress pituitary or ovarian function, bringing on menstrual disorders, failure of estrogenic and luteal functions, or anovulation.

b. The excess circulating androgens from adrenal tumors are thought to disrupt the ovario-endometrial-pituitary reciprocities, either by direct depression of the estrogenic metabolism or by damage of gonadotropic activity. It is also believed that a gametopathic defect is imposed.

3. *Pancreas:* Just what part the pancreas plays in reproductive inter-relations is not known, but the fact that diabetes mellitus often is accompanied by retardation of adolescence, menstrual irregularity and sterility, makes probable the explanation that general constitutional depression affecting adversely the ovary and pituitary metabolisms, is the solution. Almost certainly a gametopathic factor is present, for insulin therapy of diabetic females frequently is found to raise the incidence of pregnancy without affecting the rather high rate of abortions prevalent in these women. In older cases of diabetes early sclerosis of ovarian vessels may operate to shorten the ovary's active life.

The general governing action of the pituitary, the secretory functions of the ovary, and the reciprocal relation between them are too well known to dwell upon. It is probable that inadequacy of the anterior pituitary during the taxing period of adolescence is responsible for most endocrinopathies leading to sterility. By the time the adult patient is seen, the pituitary (if not diseased) has recovered its normal functional adequacy, leaving as residuals hypoplastic ovaries and damaged capacities in other glands.

Causes of anovulation are made up of constitutional endocrine, non-endocrine, and some local factors mechanically preventing ovulation:

I. Non-endocrine causes, acting to depress the metabolism of the ovary, the pituitary, or both, by general constitutional impairment:

A. Dietary errors: Deficiencies of protein and of vitamins B and C; excess leading to obesity.

B. Cachexia from chronic infectious or other debilitating disease.

C. Acute infections.

D. Chronic intoxications.

E. Changes in altitude and climate.

F. Anemias and blood dyscrasias.

G. Emotional crises and psychic disorders.

II. Non-endocrine local causes:

A. Ovarian: Congenital anomalies such as aplasia or intrinsic germinal inadequacy; damage by radium, x-ray, surgery, or torsion; polycystic condition caused by fibrotic tunica albuginea; fibrosis and scarring due to pelvic inflammatory disease, endometriosis, or neoplastic change; persistent corpora lutea and uterine and adnexal displacements compromising ovarian blood supply, adnexitis, and old adnexal pregnancies.

B. Pituitary: Intrinsic germinal inadequacy, damage by surgery or radiations; tumors, abscesses, and vascular accidents of an embolic, thrombotic or hemorrhagic nature.

III. Endocrine causes of anovulation:

A. The causes of primary ovarian failure:

1. Non-endocrine constitutional causes already described.

2. Local factors affecting the ovary, already listed.

3. Germinal fault or fetal disease of the ovary.

4. Incomplete puerperal recovery, in late type primary failure.

5. Disordered thyroid, pancreatic, or adrenal function, (which have been mentioned), occurring especially prepubertally.

6. Adolescent pituitary failure, with later pituitary recovery, leaving unresponsive ovaries.

7. Adrenal cell rest (virilizing) tumors of adrenal cortex and ovary, arrhenoblastoma of ovary; and granulosa and theca cell ovarian tumors leading to hyperestrogenism, menstrual irregularity and anovulation.

8. Endometriopathic factors may primarily affect the pituitary, or upset ovariopituitary reciprocities. Patients may secrete pregnandiol and still bleed from estrogenic endometrium; may give no progestational response to cyclic substitutional progesterone therapy, and may fail to respond to gonadotropic therapy until uterine hyperplasia and endometrial proliferation have markedly stimulated by estrogenic therapy.

9. Hyperpituitarism causing polycystic ovary, with virilizing effects of androgen imbalance.

10. The common causes of early primary ovarian failure are:

a. Intrinsically germinal.

b. Childhood pituitary deficiency.

c. Childhood hypothyroidism.

d. Inanition from inadequate diet, or cachexia from acute or chronic illness.

B. Causes of secondary ovarian failure:

1. The non-endocrine local factors affecting the pituitary.

2. Constitutional causes affecting the pituitary.

3. Intrinsic germinal or developmental fault in the pituitary usually manifested by its failure during periods exerting great demand as at puberty or pregnancy.

4. Pancreatic, thyroid, or adrenal disorder depressing local metabolism of pituitary.

5. Endometriopathic factors, already described.

IV. Gametopathic failure: According to Moench "Defects in either one of the two germ cells lead not only to sterility but in lesser degrees to abortion, premature labors, still-births, and fetal anomalies." Besides the general causes of anovulation, most of which are capable of imposing a gametopathic failure, there are some conditions especially notable therefor: diabetes, hypothyroidism, germinal defects, radiation injury, virilizing tumors, and grades of hypopituitary function not producing anovulation.

V. Luteal failure: The secretion of progesterone may be deficient in amount or in duration ("short luteal phase"). It is caused by essentially the same factors bringing on anovulation, and is part of the latter condition when it occurs. If there is normal ovulation followed by luteal deficiency, there occurs no nidation or poor nidation and death of the ovum.

In the diagnosis of ovarian failure, we are here chiefly concerned with identifying the condition as either primary or secondary, since choice of therapy will be so determined. Consideration will be given in detail only to those features of a case that lead to a diagnosis of one or the other condition. The diagnostic schedule that should be used in every sterility investigation will uncover these causes and indicate the therapy therefor.

Before launching into an account of the minimal requirements for an inquiry into the etiology of sterility, certain principles of diagnosis should be borne in mind.

1. Meaker states "There is no worse fallacy than to accept the first discovered abnormality as the sole cause in the case under consideration, on the unjustifiable assumption that no other causative factor is present."

2. It is essential to be assured of an ade-

quate grade of male fertility before subjecting a wife to any prolonged diagnostic or therapeutic procedure. This does not imply the omission of routine history, physical examination, and laboratory work on the female until the male is cleared.

3. The complaint of sterility in itself suggests endocrine disorder, but except in obvious cases of endocrine disaster the question of such causes arises after a thorough examination of both partners has failed to reveal a structural or major constitutional disorder in either partner.

4. The purpose of the diagnostic study is not only to discover why the marriage is barren, but also whether a pregnancy is desirable from considerations of the mother's health and the possibility of producing a normal, viable child. If during an investigation it should be found that there are present inadequately treated lues in either mate, heritable and constitutional taints, advanced and severe hormonal disorder not likely to be curable, or grave renal, cardiac, or pulmonary disease in the wife, the wisest course is to inform the couple of your opinion and abandon the investigation.

In accordance with these principles it is recommended that the male be eliminated first as a source of the sterility unless some glaring reproductive fault of the female is discovered on routine physical examination. This is accomplished by establishing that the husband is normal in the following respects:

1. History and physical establish presence of potentia and libido.

2. Blood count, serology, urinalysis, basal metabolic rate determination, all within normal limits.

3. The examination of a fresh seminal specimen for volume, motility, count, and abnormal sperm forms; this is supplemented by the Huhner post-coital examination of the wife's endocervical secretions, serving to establish delivery of sperm to the cervix, and sperm motility and viability in endocervical mucus. These two tests constitute the most significant portion of the husband's examination.

Having absolved the latter of any major defect, the investigator next undertakes the following procedures to discover whatever abnormalities are present in the wife:

1. Complete blood count, urinalysis, serology, basal metabolic rate determination. With regard to the latter it is pertinent that very low grades of hypothyroidism can depress ovarian function, and that most of the errors in technic of basal metabolic rate determination tend to raise the reading.

2. Complete physical examination, including a careful gynecologic survey of:

A. Tubal patency, by insufflation with CO_2, or oxidized oil and subsequent roentgenography; or many physicians merely use air insufflation.

B. Patency of cervix to sperm as judged by Huhner's test; five to twenty normal, motile sperm in the cervix indicate good delivery and reception.

C. Endometrial biopsy, best performed within six to eighteen hours after onset of menstrual flow. If an estrogenic or immature progestational endometrium is found under microscopic examination, several more cycles should be studied, with the aid of urinary pregnandiol and basal rectal temperature methods to determine whether the cycles are habitually anovulatory.

Despite all the subjective symptoms (mid-cycle uterine bleeding and/or mittelschmerz) and objective tests of the time of ovulation, there is still no way of knowing definitely whether the release of fertilizable ova occurs. Some of the methods that have been shown to be fairly reliable are the following:

a. Basal rectal temperature records (showing a drop, then a rise, in daily determinations made throughout the cycle, occurring usually 12 to 14 days before onset of next menstrual flow).

b. Endometrial biopsy and examination (showing secretory, progestin stage endometrium).

c. Potential changes between symphysis and vagina.

d. Vaginal smear and pH of vaginal secretions.

e. Double peak of excretion of estrogen and F. S. H.

f. Increase in amount and decrease in viscosity of endocervical secretions.

4. Further endocrine studies, if primary hypoovarianism is suspected:

a. X-ray films of the sella turcica to rule out pituitary overgrowth or local damage.

b. Determination of upper and lower measurements (crown to symphysis pubis, and symphysis to sole, respectively). Measurement of height and span.

c. X-ray films of the epiphyses of the long bones to determine the state of epiphyseal closure; delayed closure seen in hypothyroidism and hypoovarianism, as well as in other conditions. This test and visualization of ossification centers are of little value in most sterility investigations.

d. Urinary levels of gonadotropic and ovarian secretions are difficult to determine and rarely practicable under ordinary conditions.

The actual diagnosis of failure of ovulation is made by the finding of proliferative or poorly secretory endometrium on biopsy at about six to 18 hours after onset of the flow, in several cycles. That this method is fallible and needs support by the complementary method of basal rectal temperature graphs is shown by these considerations:

1. Corpus luteum can form without liberation of the ovum.

2. It can fail to form after ovulation.

3. Secretion of progesterone may be too brief or insufficient in amount.

4. The endometrium may be refractory.

"A history of amenorrhea, oligomenorrhea and irregular catamenia should arouse suspicion of anovular menstruation, sufficient to be an indication for repeated endometrial biopsies in succeeding cycles."

Once the diagnosis of anovulation has been made, the rest of the management of the case depends upon the modifying diagnosis, primary or secondary ovarian failure. For diagnostic purposes the anovulation of primary ovarian failure differs from that of the secondary type in that the former is accompanied by most of the stigmata of either arrested puberty or premature senility or menopause. If it be of the early or adolescent kind the findings will be as follows:

I. Early primary ovarian failure:

A. Minimal or no sexual maturation.

1. Internal and external genitalia remain infantile; cervix is long and conical, uterine index is less than .60 (index equals half of body length minus cervix length, average value .75).

2. Breasts are about neutral stage, and other local fat pads and crines fail to appear.

3. Menarche late or absent.

B. Disorders of bone growth; span exceeds height, lower measurement greater than upper; maturation of epiphyses and ossification centers either late or absent.

C. There is absolute sterility.

D. Endometrial biopsy shows a hypoestrogenic condition; vaginal smear and pH determination reveal a poorly cornified epithelium and a pH above normal.

E. Urinary gonadotropins are normal or elevated; urinary estrogens depressed. It is thought that when the ovary fails, the checking mechanism of the anterior pituitary is gone, so that the output of F. S. H. becomes excessive. This would seem especially true of late primary failure.

II. Late primary failure: This condition is characterized by regression and loss rather than failure of maturation of sex organs. The other two signs of primary ovarian failure, amenorrhea and absolute sterility, are prominent features of these cases.

A. Onset is during the reproductive age, and mimics the climacteric.

B. Endometrial biopsy reveals a thin, atrophic lining of the uterus.

C. There is anovulation.

D. Ovaries shrunken and sclerotic.

E. Vaginal smear shows poor cornification.

F. Regression of cervix and uterus, although not to the extent of that in early primary ovarian failure.

G. Trial with gonadotropic therapy evokes no response.

H. Urinary gonadotropins are elevated, and the estrogens are depressed.

III. Secondary ovarian failure:

A. This condition may have only sterility, or metro-menorrhagia as the chief complaint.

B. The patient is well feminized and normally developed, showing no outward evidence of endocrine disease.

C. If extra-ovarian endocrine or non-endocrine diseases exist, their symptoms will appear, for example, Addison's disease, acromegaly.

D. Basal metabolic rate is often found to be slightly depressed. (This fact may have something to do with the lamentable practice of giving thyroid for any and every endocrine complaint or menstrual disorder.)

E. Obesity of the girdle type is a frequent finding.

F. Absence of estrogens and gonadotropins in the urine (in primary pituitary failure).

G. Or there may be high estrogen titer and no gonadotropins found in the urine, or usually, some gonadotropic activity is found, but no pre-ovulatory peak of F. S. H. excretion. In general, pregnandiol studies are not reliable.

H. A tendency to small extremities with fragile bony structure.

I. Late calcification of ossification centers.

J. Sometimes a high sugar tolerance.

K. The best diagnostic criterion is the finding by biopsy of estrogenic or poor progestational endometrium at the onset of two consecutive episodes of bleeding. In general, as previously indicated, this condition consists of abnormal uterine bleeding and sterility not associated with evidences of estrogenic failure, local gynecologic pathology, or general disease or physical impairment.

Before outlining specific therapies for these several conditions it is well to consider a few guiding principles, and to point out that excessive enthusiasm for any endocrine therapy is unjustified at this stage of our knowledge. Apparent cures by any method are always subject to the criticism that they might be spontaneous; for example, a woman may ovulate spontaneously after several anovulatory cycles. In general, the best results are obtained by physiologic therapy: the use of androgens would seem to have no logical place in the treatment of sterility of females. So-called stimulating doses of x-ray to the ovaries and pituitary, although well championed by Kaplan and others, is generally considered to lead ultimately to more profound ovarian failure.

In the use of endocrine therapy the dictum, "No endocrine gland is stimulated by products which it, itself, produces," is a warning against injudicious deluging of the reproductive system with hormone preparations. Investigating the current practice of administering thyroid preparations indiscriminately in the therapy of all sterility, Winkelstein states, "The effect of thyroid as an adjuvant or curative measure was only demonstrated where lack of thyroid was present." Before plunging into substitutional or stimulative endocrine therapy, it is the wiser course first to give adequate treatment to extra-endocrine constitutional and local disorders contributing to sterility. Again, if hypoestrogenism or hypopituitarism coexists with disease of the thyroid, adrenal or pancreas, priority of treatment is granted to the latter three conditions. With the foregoing reservations in mind, then, the following suggestions as to therapy of primary and secondary ovarian failure are presented:

A. Treatment of primary ovarian failure:

With the possible exceptions of cases due to pelvic inflammatory disease, endocrine tumors of the ovary, polycystic ovary, and occasional delayed maturation or delayed recovery from puerperal depression, the ovary of primary failure does not respond to any therapy by complete functional salvage (resumption of cyclic bleeding, spontaneous recurrence of normal ovario-endometrial relations, and occurrence of pregnancy after cessation of therapy). There are two courses of therapy open, usually employed sequentially. The first of these is

a trial with the "one or two cyclic gonadotropic" treatments described by Hamblen. If there is no response to the trial, "priming" is attempted, using estrogen and progesterone in cyclic fashion, alternating with courses of gonadotropins. Here the rationale is the possibility of an endometriopathic condition, which may allow the ovary to regain its responsive power once the disordered ovario-endometrial system is righted. Finally, if it is evident that salvage will not be obtained, substitutional therapy is undertaken for the cosmetic and psychic benefits to be derived.

The dosages used are:

1. Gonadotropic trial: Day five through day 14 give daily intramuscular injections of 400 U of P. M. S., having first tested for sensitivity to serum. Day 15 through day 24 give daily intramuscular injections of 500 IU of chorionic gonadotropins (containing L.H.). If there is bleeding during the treatments the injections are stopped; a biopsy is then performed within 12-18 hours; a progestational endometrium indicates a specific response.

2. Priming doses: Estrone sulfate, 3.75 mg. per day orally, day five through day 25; and progesterone, 10 mg. intramuscularly daily from day 15 through day 25. This is continued until the occurrence of regular menstruation and a significant degree of uterine hyperplasia is obtained; then the trial of gonadotropic cyclic dosage is used as in "1" above.

3. When these fail, as they usually will, replacement therapy is instituted. This may close the epiphyses and accomplish some secondary sex character maturation, to the patient's considerable satisfaction. If some uterine growth is thus achieved, the response of the ovary to gonadotropins may still change; the dosages are the same as those used for priming.

B. Treatment of secondary ovarian failure:

As far as conception is concerned this is the most hopeful type. Ovulation is accomplished, when successful, by the use of the "one or two cyclic gonadotropic" schedule, with the same dosage as above. The restoration of ovulation and luteal function is rarely more than temporary; the treatment is onerous and expensive. For these reasons gonadotropic therapy is usually limited to attempts at pregnancy. It is not given for more than two cycles consecutively, allowing for two or three months rest periods between treatment series to avoid the production of antibodies to the foreign protein in the P. M. S.; for the latter reason also the patient is skin-tested for sensitivity before each series.

If the fault is endometriopathic, the patient is maintained on estrogen and progesterone substitution dosage, and occasional gonadotropic trials at pregnancy are employed as previously described. Actually, the use of any such dosage figures as have here been recommended is a stab in the dark, for every patient has different requirements. This therapy should be discontinued as soon as it becomes apparent that no response is forthcoming, remembering that glands can be depressed by the administration of their own secretions. It is claimbed by Hamblen that 51 per cent of young women with this type of anovulatory failure yielded specific, although temporary, responses to the gonadotropic therapy described. If there is no response it is recommended that a cycle be skipped and therapy started in the next cycle using 1000 I U of the gonadotropes as a daily dosage. Should such strong measures "force" a pregnancy, there is found a marked tendency to early abortion, which must be circumvented by adequate doses of progesterone (10) mg. three times a week) until past the point of viability of the fetus. Some authors prescribe the simultaneous use of 10,000 rat units per day of estradiol benzoate, three times weekly.

In a review of five years' results with gonadotropic therapy, Hamblen and Davis report achievement of pregnancies in 20.3 per cent of their patients suffering from ovarian sterility.

Sterility resulting from early or late primary ovarian failure has a very poor outlook for salvage, but it is in these condi-

tions that prophylaxis can play a great part. Much of the early type is preventable by proper attention to the menstrual behavior and endocrine and constitutional status of the girl, since a slight deficiency later becoming a major threat to fertility can often be easily managed during adolescence. Other measures helpful in avoiding late primary failure are the teaching of intelligent sex hygiene, control of venereal disease, premarital advice, including practical instruction in contraceptive methods, and the avoidance of all unnecessary pelvic operations. Although there is a great deal of value in attention to such factors, by far the most arresting possibilities are in the endocrine aspects of sterility.

For several centuries this absorbing quest into the nature and manner of action of the hormones involved in fertility has made its halting way by dint of the thousands of small but important contributions of individual workers in the basic sciences, clinical medicine, gynecology and endocrine chemistry; laborious extractions of secretions from animal sources gives way little by little to laboratory syntheses of potent imitations of the original product. During the last 25 years the progress has been tremendous, but the frontier for research in endocrinology shows no signs of disappearing; it recedes and broadens with every new discovery.

Space would not permit the publishing of over 150 references to this article; any one interested in consulting the bibliography may obtain it from the office of the journal.

I wish to express my gratitude to Dr. B. B. Weinstein for his invaluable aid in selection of literature and suggestions for the plan of this paper.

NEW ORLEANS

Medical and Surgical Journal

Established 1844

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THE AMERICAN MEDICAL ASSOCIATION MEETING IN ATLANTIC CITY

It is said that the recent meeting of the A. M. A. held the first part of June was the largest that has ever been held by the organization. The final figures were unobtainable when we left this seashore city but the list of registrants was still behind time in the publication of the Association's daily bulletin. The convention in many ways was comparable to a three ring circus. There was the enormous crowd, there were innumerable activities and e v e r y b o d y seemed to be thrilled and excited about the whole thing.

The opening two days proved very profitable and were well liked by the attendants. There were hour or less talks delivered by prominent doctors both from this country and from abroad. The list of distinguished guests from other nations was quite impressive.

The scientific assembly as per usual was extremely well attended and always there seemed to be large crowds about the more important exhibits. The scientific assembly is well worth a trip to any meeting of the A. M. A. Nowhere that I know of is there such a concentrated mass of scientific material made readily interpretable and with the exhibitors prepared to answer questions or to make clear any detail that might not be entirely lucid.

A word must be said about the commercial exhibits. It is the commercial exhibits that help to meet the enormous expenses that necessarily occur with this meeting. These exhibits are well worth while. The physician can browse amongst the newest books of medical publishers throughout the country; he can learn about the latest in drug therapy; he can see the remarkable scientific advances made by some of the pharmaceutical houses and food manufacturers; and he can view the very latest in the field of surgical instruments.

The regular section meetings held the latter part of the week were also remarkably well attended. The enormous lecture hall in the largest auditorium in the United States which was occupied by the Section on Medicine was filled with each meeting. Another very popular feature was the panel discussion of certain aspects of diseases, their cure and their management in general. They were just about as popular as any one feature of the meeting.

All in all it well repays a man to attend the annual session of the A. M. A. This be-

ing the centennial year probably there were a few more attendants than there might otherwise have been but nevertheless the session undoubtedly would have been very large. Meetings are getting so big that there are only one or two cities that can hold them conveniently. Next year the annual meeting will be held in Chicago, the following year the organization will return to Atlantic City and the succeeding year it will be on the Pacific Coast, either San Francisco or Los Angeles whose citizens are promising to build a large auditorium.

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A. M. A. HONORS LOUISIANIANS

The far South and the State of Louisiana have reason to feel proud of two of its medical profession. Both of these doctors have lived all their lives, until they completed their medical course, not far from the banks of the Mississippi and they have both of them the traditions of the Creoles. It just so happens that the proof of the first statement lies in the fact that the two highest honors for scientific work exhibited at the A. M. A. meeting were given to Dr. George E. Burch who received the Gold Medal and Dr. Guy Faget to whom was awarded the Silver Medal, the second prize. Dr. Burch is the son of a well known country doctor who for many years practiced at Edgard, Louisiana. Burch is a typical local product, in fact up until the time he graduated or shortly thereafter he had never been out of the State of Louisiana. The second award was given to Dr. Faget, another member of a family that has been well known in medicine for many years. Dr. Faget, in collaboration with Dr. Erickson, had a perfectly beautiful exhibit of the effect of the sulfones in the treatment of the leprous patient. Dr. Faget's family is steeped in medical tradition. One of his grandfathers was a doctor, as was an uncle. He was one of three boys who entered Tulane Medical School many years ago and graduated with excellent records. The three Doctors Faget all entered the Public Health Service, two of them are still in the service, one has resigned and is practicing in New Orleans.

We want to congratulate our two local representatives on obtaining the two highest honors in the scientific exhibit. It may not be inappropriate to add that of the four honorable mentions, one of them was given to the exhibit of Dr. Champ Lyons and his group at Tulane.

It might give pride to Tulane Alumni to know that both Burch and Faget are graduates of Tulane Medical School.

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NEW PROFESSOR OF SURGERY AT L. S. U.

We are extremely sorry to hear of the retirement of Dr. Urban Maes as Professor of Surgery and head of the Department of Surgery at L. S. U. Dr. Maes has been appointed Professor Emeritus. We are delighted indeed with the selection of Dr. James D. Rives as Professor and head of the Department of Surgery to succeed Dr. Maes. Dr. Rives is very well known amongst our local medical profession for his surgical accomplishments. He is a man of charm and highest intelligence who has always been willing to help out a brother practitioner in distress. We look forward to a highly successful term of office for this outstanding surgeon and surgical investigator.

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MEDICAL USE OF RADIOACTIVE ISOTOPES

In the review section of the American Journal of Medical Sciences there is an excellent article[1] on the medical use of radioactive isotopes in hematologic disturbances and neoplasms. It is far from the intention of the writer of this editorial to suggest that he knows very much about radioactive isotopes. An isotope refers to forms of the same element having different atomic weights. It is almost unbelievable and beyond the imagination of man that neutrons can be forced into nuclei of some atoms, producing a new atomic weight for the material and yet this particular element is chemically identical with its prototype that is not radioactive. The radioactive isotope decays with a varying degree of rapidity

and the figures are astronomical. In the instance of radiophosphorus, 35 of every million atoms are changed to stable sulfur spontaneously each minute. Some of the periods of "half life" are in seconds, others are in the terms of innumerable decades. The atom is, needless to state, extremely small but radioactivity of an isotope in terms of millicuries is again in figures almost unbelievable. A millicurie is the amount of radioactivity that has been produced by disintegration of 37 million atoms per second while one microcurie is one one-thousandth of one millicurie.

In the treatment of disease of the blood and hematopoietic system great hopes were held that this radioactive material might be of tremendous help in curing all diseases in which the bone and bone marrow are primarily affected. Unfortunately this has not been the case. The one disease that has yielded most favorably is polycythemia vera but after all this is a disease which is relatively rare and because of its rarity is of no particular significance or importance. In leukemia the results have not been particularly satisfactory. The isotope is somewhat easier to take than radiation therapy and in some respects might prove a useful adjunct to the treatment with x-ray, but on the whole the tremendous furor which was created a few years ago about this remarkable substance which would cure leukemia, Hodgkin's disease and lymphoblastoma has not eventuated, so that once again we are placed in a position where a therapeutic agent was popularized to too great an extent by the press. Hope was held out to the sufferers of leukemia if they could just get hold of some radiophosphorus the disease would be cured. Such has not been the case to the sorrow and disappointment of innumerable sufferers.

The second very short review has to do with radio-iodine and the thyroid by F. Raymond Keating, Jr.[2] Here the isotope has proved of particular value in studying the normal and the abnormal thyroid gland but when radioactive iodine was first brought out it was felt that it would be possible to control without difficulty individuals who have cancer of the thyroid gland. Unfortunately this has not been substantiated after careful clinical trial. On the other hand, it is interesting to note that in a very few instances the effect has been not on the thyroid gland itself but on the distant metastases, more particularly the bony metastatic lesions. This form of treatment is certainly sufficiently suggestive to warrant a continuation of the use of radio-iodine in malignant inoperable lesions of the thyroid.

1. Hall, B. E., and Watkins, C. H.: The medical use of radioactive isotopes. I. Radioactive isotopes in hematologic disturbances and neoplasms, Am. J. Med. Sci., 213:621, 1947.

2. Keating, F. Raymond, Jr.: The medical use of radioactive isotopes. II. Radio-iodine and the thyroid, Am. J. Med. Sci., 213:628, 1947.

ORGANIZATION SECTION

The Executive Committee dedicates this section to the members of the Louisiana State Medical Society, feeling that a proper discussion of salient issues will contribute to the understanding and fortification of our Society.

An informed profession should be a wise one.

SENATE BILL 545

Immediately following the 1947 meeting of the House of Delegates of the State Society the President, Dr. Gilbert C. Anderson, appointed a special committee composed of Dr. J. P. Sanders, Chairman, and Drs. Guy R. Jones and W. P. D. Tilly, to prepare a brief on Senate Bill 545 for presentation at the hearings on this bill in Washington. Such a brief was submitted to the Executive Committee on May 31 and after discussion of the matter as presented, the special committee with the chairman of the Congressional Committee, the first vice-president, in the absence of the president, and the secretary, revised the brief to meet the wishes of the executive Committee. This brief, in its final form, is printed in

this section of the Journal so that all members of the Society may be familiar with the stand the organization has taken in reference to this legislation. It is felt that the special committee presented the bill in an excellent manner to the Committee on Labor and Public Welfare, before whom the hearings were held, and comment carried in the bulletin from the Council on Medical Service of the American Medical Association concerning this appearance will be found immediately following copy of the brief.

BRIEF BY THE LOUISIANA STATE MEDICAL SOCIETY BEFORE THE COMMITTEE ON LABOR AND PUBLIC WELFARE UNITED STATES SENATE HEARINGS ON S. 545

NATIONAL HEALTH BILL

Submitted by
DR. GUY R. JONES
DR. W. P. D. TILLY
DR. J. P. SANDERS
June 6, 1947

OUR STAND TOWARD THE PRESENT LEGISLATION, S. 545

Senate Bill 545 concentrates all the health activities of the Federal Government under one head. We believe that this is an important change because it co-ordinates all the activities and prevents duplication of efforts. It will produce economy in the long run and will be responsible for the activities in one department. We believe that health is so important that the *Administrator should be on a cabinet level.* We thoroughly agree that the Administrator should be a native born citizen of the United States, who is a practitioner of allopathic medicine, with an experience of at least five years practice in a rural community or town of five thousand or less. We believe that a general practitioner gets closer to his patients than the specialist and, consequently, would have a greater understanding of the needs of the people. We believe that the Surgeon General of the Public Health Service, the Directors of the Medical and Hospital Care Services, Maternal and Child Welfare Services should also be similarly qualified. We strongly recommend that the medical care service be kept in the hands of the local communities to be served. The communities, including their doctors, know the communities' needs. Many of them are working on these needs and with a little help, properly placed, can solve their own problems better than they can be solved by anyone else in the world. In the event of disagreement between the local community and state agency, appeal can be made by the community to the Administrator.

LOUISIANA'S CONTRIBUTION TO MEDICAL CARE

This brief on Senate Bill 545, the National Health Bill, is presented for the Louisiana State Medical Society, representing a total membership of 1,659 doctors out of a total of 2,507 registered in the State of Louisiana. We are responsible for the medical care of the people of the State of Louisiana which has a population of around two and one-half million people.

Louisiana has contributed to a large extent to the present status of the high standard of medical practice in the United States. It is the home of two great medical schools, Tulane University Medical School and Louisiana State University Medical School. These two schools have trained some of the great medical men of our time. Louisiana has a total of 167 hospitals, both general and special, with a bed capactiy of 9,227.

Our State Medical Society was organized sixty-seven years ago and has constantly stood for the highest type of medical practice. Through its medical schools, hospitals, medical conventions and societies, Louisiana has tried to train its doctors on the highest plane possible. Through these same organizations we have tried to keep our skirts clear of many of the isms that have crept into other states. As time has progressed we have tried to improve accordingly. We have cooperated with the Government and many other lay groups in keeping the practice of medicine free. We believe that patients get better treatment under the American plan of the present doctor-patient relationship than under any other system in the world. We are willing to vary that plan by the trial and error method whenever it seems feasible to do so.

LOUISIANA IS ATTEMPTING TO GIVE GOOD MEDICAL CARE TO ALL ITS PEOPLE

Economically people fall in three general classes so far as medical care is concerned:

1. The well-to-do or rich.
2. The marginal group.
3. The indigent.

The first group of people, the well-to-do or rich, are financially able to take care of themselves and, therefore, need no help or guidance.

The third group, the indigent, is very well taken care of in Louisiana through our system of Charity Hospitals, Charity Clinics, Department of Public Welfare and many other social agencies. Louisiana has seven Charity Hospitals with a bed capacity of 4,808 for care of indigent alone. There are additional beds for indigents in private hospitals throughout the state. These Charity Hospitals are strategically situated in different portions of the state. They are in close enough proximity that most any indigent person can be carried there in a short period of time. These hospitals are well equipped with x-ray, clinical and pathological laboratories, physiotherapy department, nursing

schools, graduate nurses and technicians and a well trained medical staff that can take care of any of their medical needs.

The second group, the marginal group, is the one about which we are greatly concerned. We must remember that many of our great leaders have come and are coming from this marginal group. In our eagerness to help them solve their problems we should not deprive them of the initiative that may later make them leaders in industry, the professions, or in politics. We must give such help in such a way that it will stimulate their initiatives but will not hamper their progress. The marginal group to a large extent covers four classes of people:

1. Most all farmers, particularly the small operators.
2. Large families who have moderate incomes.
3. Small families with moderate incomes who have unusual responsibility for some reason or other.
4. Those with definite low incomes because of their own inefficiency or otherwise.

Most individuals in this group are very independent. They want the better things of life but they want to pay for them. They detest charity. The farmers in particular are the most independent people in the world and that largely is the reason they are farmers even in spite of their low income. The farmers are developing cooperatives, both producers and consumers, in order to help themselves. These include medical services in many instances. Hospitalization insurance, particularly the Blue Cross plan, is well developed in Louisiana. The low income groups are particularly interested in this type of insurance to take care of those catastrophic illnesses which all too often ruin the marginal family.

The Louisiana State Medical Society has been working on a prepayment medical service for three years and was able to start issuing policies in November, 1946. The Louisiana Physicians Service is the insurance company organized and operated by the Louisiana State Medical Society. To date (seven months) over 15,000 policies have been sold in groups alone. The policy is planned to take care of catastrophic illnesses such as surgery, obstetrics and certain types of laboratory and x-ray examinations. Later we hope to take care of all the medical care of policy holders. Over seventy-five per cent of all the doctors in Louisiana have already agreed to take care of the marginal group for the nominal fee provded in this policy. The people are cooperating in this prepayment medical plan. It is the American way of solving an American problem.

The Louisiana State Medical Society has a very active Committee on Rural Medical Service. It is composed of five physicians located in different portions of the state. At our state convention held in New Orleans four weeks ago this committee was given broad powers to cooperate with the Farm Bureau, Rural Hospital Service and other interested groups to improve rural medical care in the State of Louisiana. We are encouraging general practitioners to settle in rural areas. We are working with the State Hospital Council in locating rural hospitals at strategic areas in the rural communities as provided for under the Hill-Burton Bill. The Rural Health Committee of Louisiana is anxious to see rural people obtain just as adequate medical care as the people of urban areas.

MEDICAL CARE IS EXPENSIVE

The cost of good medical care has become expensive not because the doctors want it but because the patient demands it. The patient is not satisfied to go into a doctor's office for him to take his blood pressure, temperature, look at his throat, listen to his heart and write one or two prescriptions and let him go. The average patient would say that this is poor medical care. The average patient who now goes to a doctor for a complete checkup requires:

1. A complete medical history.
2. A complete physical examination.
3. Laboratory examinations whenever necessary of the blood, urine, stool, secretions of the body, blood chemistry and many special examinations that may be required.
4. An x-ray examination of any or all parts of the body that might be involved.
5. Heart tracings are frequently demanded by patients who have high blood pressure or heart disease.
6. Special eye, ear, nose, throat, kidney, stomach, rectal and other examinations may be required.

All of this means that the patient has increased the expense of his examination enormously from that which was required fifty years ago. First of all, the physician has to have a well equipped x-ray and clinical laboratory with trained technicians and nurses to operate them. More space is required and more clerical help is necessary. The treatment of the patient's condition may not be so expensive after he has been diagnosed. However, many cases may require infusions, transfusions, plasma and other expensive drugs not formerly known such as penicillin, streptomycin, et cetera. We might as well try to get our patients to go back to the horse and buggy days for their transportation as to take them back for cheap medical care. The horse and buggy cost approximately $200.000, the old Model T could be bought for around $600.00 while the present automobile will cost around $1,500.00. People in the marginal group, however, require an automobile in which to ride in spite of the cost and also they want modern medical care regardless of the increased cost. None are willing to go back to the horse and buggy days.

Medical practice in this Country has progressed to the present high standard under the American plan. Whenever any attempt is made to regiment medical practice it deteriorates.

RECOMMENDATIONS

1. That the Administrator should be a native born citizen of the United States, who is a practitioner of allopathic medicine with an experience of at least five years practice in a rural community or town of five thousand or less.
2. That the qualifications for the Surgeon General of the Public Health Service, the Directors of the Medical and Hospital Care Services, Maternal and Child Welfare shou'd be the same as those of the Administrator.
3. That any appropriation under this bill be a grant-in-aid on a community level.

CONCLUSION

We recognize Senate Bill 545, with alterations as suggested in this brief, to be the most favorable bill presently offered for the American people and the medical profession.

Excerpt from Bulletin No. 8 of the Council on Medical Service on the American Medical Association

"There came from the Louisiana State Medical Society Doctors Guy R. Jones, W. P. D. Tilly and J. P. Sanders, who stated that in their opinion this bill was the best bill presented in recent years, both to the public and to the medical profession.

"They offered several suggestions for changes in the Bill particularly in its administration. They suggested that the administrator of the Act and the Directors of the different departments should be practicing physicians and experienced in general practice. They stated that the general practitioner was closer to the people and knew their needs better than men in the specialties. They said that any grants should be on a community basis and that the Administration, both community and state, should be in the hands of laymen and doctors.

"They observed that this Bill was intended to take care of that large group of people of marginal income. It was pointed out that many leaders in all walks of life came from this group and that any assistance given them should be done in such a way that it would not hamper their initiative or retard their progress."

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

July 14, Joint Scientific and Second Quarterly Executive Meeting, Orleans Parish Medical Society, 8 p. m.

July 17, Medical Toastmasters, Hutchinson Memorial Auditorium, 8 p. m.

July 18, Lakeshore Hospital Staff, 8 p. m.

July 23, Catholic Physicians' Guild, 8 p. m.

July 24, Clinico-pathologic Conference, Touro Infirmary, 12 noon.
DePaul Sanitarium Staff, 8 p. m.
Medical Toastmasters, Hutchinson Memorial Auditorium, 8 p. m.

July 25, New Orleans Hospital Dispensary for Women and Children Staff, 8 p. m.

Aug. 4, Board of Directors, Orleans Parish Medical Society, 8 p. m.

Aug. 7, Clinico-pathologic Conference, Touro Infirmary, 12 noon.
Executive Committee, Baptist Hospital, 8 p. m.
Medical Toastmasters, Hutchinson Memorial Auditorium, 8 p. m.

NEWS ITEMS

Dr. Howard Mahorner addressed the Birmingham Surgical Society in Birmingham on May 26. He spoke on the surgical significance of duodenal diverticula.

Dr. John H. Musser was recently re-elected (for the twelfth time) president of the Child Welfare and Community Health Association.

Dr. Rudolph Matas was the principal speaker at the Paul Tulane Day Ceremonies at McAlister Auditorium, May 10.

Highlight of the program was the presentation of a plaque commemorating the occasion to Dr. Matas, one of the oldest living graduates of Tulane University.

Dr. Lucy Scott Hill and Dr. Sam Nelken were elected to membership in the American Psychiatric Association at its 103rd annual meeting in New York City recently.

LOUISIANA HOSPITAL ASSOCIATION

Dr. Lewis E. Jarrett was elected president of the Louisiana Hospital Association at a meeting of the Association in Lafayette, May 24.

CHANGES IN MEMBERSHIP ROLLS

During the month—

Two physicians were separated from military service—Dr. Philip B. Johnson and Dr. Albert P. Spaar, Jr. Dr. Johnson was removed and Dr. Spaar was transferred to intern membership.

Four physicians were dropped because of nonpayment of dues. (three active—Drs. John M. Carter, J. W. Rosenthal, and Marvin C. Smith; one associate—Dr. Robert P. Vincent).

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

Society	Date	Place
East Baton Rouge	Second Wednesday of every month	Baton Rouge
Morehouse	Second Tuesday of every month	Bastrop
Orleans	Second Monday of every month	New Orleans
Ouachita	First Thursday of very month	Monroe
Rapides	First Monday of every month	Alexandria
Sabine	First Wednesday of every month	
Second District	Third Thursday of every month	
Shreveport	First Tuesday of every month	Shreveport
Vernon	First Thursday of every month	

LOUISIANA STATE UNIVERSITY

The School of Medicine, Louisiana State University, announces the following appointments:

James D. Rives, formerly clinical professor of surgery, as professor and head of the Department of Surgery. Rives succeeds Urban Maes, who has retired and has been appointed professor emeritus.

Robert L. Simmons, formerly director of the Lauderdale, Mississippi Health Unit, as associate professor of Public Health and Preventive Medicine on a full-time basis. Simmons succeeds George W. McCoy, who has reached the age of retirement and has been appointed professor emeritus.

Joseph A. Danna and Narcisse F. Thiberge have also reached the age of retirement and have been appointed clinical professors emeritus of surgery and medicine, respectively.

Sidney S. Chipman, formerly in the practice of pediatrics at Norwalk, Connecticut, and during the past year a student in the School of Public Health, Yale University, as associate professor of pediatrics on a full-time basis. Chipman will be director of the post graduate extension program in pediatrics in the State of Louisiana on a joint appointment of the State University and the State Board of Health.

Nelson K. Ordway, formerly instructor in pediartics, Yale University, as assistant professor of pediatrics on a full-time basis.

Edwin S. Kagy, formerly associate in Tropical Medicine at the School of Medicine, Tulane University, as clinical assistant professor of medicine.

Anthony Failla, formerly resident in otolaryngology at Charity Hospital, New Orleans, as instructor in otolaryngology on a full-time basis.

Henry C. McGill, formerly intern in pathology at Vanderbilt University Hospital, as assistant in pathology.

CHARITY HOSPITAL

A meeting of the Medical Division of Charity Hospital Visiting Staff was held on Tuesday, June 17 at 8 p. m. in the Auditorium of the Hospital. The program was as follows: Miliary Tuberculosis: Response to Streptomycin Therapy by Dr. Philip B. Johnson; Bacterial Endocarditis: Two Cases Requiring Massive Antibiotic Therapy by Drs. Norman S. Gilbert and Carl Gulotta.

OFFICERS OF LAKESHORE HOSPITAL

At a recent meeting of the staff of Lakeshore Hospital Dr. Earl Conway Smith was re-elected president. Other officers who were also re-elected include Dr. Robert M. Willoughby, vice-president; Dr. Alan Leslie, secretary-treasurer.

The members of the Executive Committee include Drs. Robert M. Willoughby, E. D. Matthews, L. J. O'Neal, W. P. Bradburn, Jr., Irvin Cahen, E. S. Nelson, J. S. Herring, W. C. Rivenbark and Harold Kearney.

AMERICAN MEDICAL ASSOCIATION MEETING

The following is a list of names of doctors from Louisiana who attended the centennial meeting of the American Medical Association in Atlantic City, June 9-13, 1947.

Alexandria: Dr. George Vryonis.
Carville: Dr. P. T. Erickson.
Franklin: Dr. D. C. Nelson.
Lockport: Dr. Guy R. Jones.
New Iberia: Dr. W. P. D. Tilly.
New Orleans: Drs. John Adriani, Ruth Aleman, J. W. Atkinson, C. A. Bahn, Elizabeth Bass, W. D. Beacham, O. W. Bethea, C. C. Blakeney, C. L. Blumstein, D. C. Browne, G. E. Burch, Edgar Burns, A. N. Caine, W. B. Clark, E. H. Countiss, O. P. Daly, J. S. D'Antoni, M. E. DeBakey, B. J. De Laureal, J. P. Farrior, V. H. Fuchs, Manuel Garcia, Grace A. Goldsmith, G. M. Haik, Katherine Havard, Lucy S. Hill, J. K. Howles, Edgar Hull, G. G. Johnson, R. A. Katz, A. S. Kiblinger, E. L. King, M. Lescale, F. L. Loria, M. G. Lynch, Champ Lyons, Rudolph Matas, Urban Maes, G. G. McHardy, J. H. Musser, Evelyn B. Nix, Alton Ochsner, W. J. Otis, William Parson, R. V. Platou, P. Pizzolato, C. T. Ray, I. L. Robbins, D. A. Roman-Vega, Dennis Rosenberg, P. J. Saleeby, D. N. Silverman, W. A. Sodeman, A. J. Sullivan,

W. G. Tabb, P. T. Talbot, G. J. Taquino, B. B. Weinstein, and G. N. Weiss.

Ponchatoula: Dr. Glenn T. Scott, Jr.

Rayne: Dr. E. C. Faulk.

Shreveport: Drs. Bodenheimer, W. H. Browning, M. D. Hargrove, D. B. Snelling, J. D. Talbot, and H. M. Trifon.

Sterlington: Dr. B. E. Spencer.

WEBSTER PARISH

The Webster Parish Medical Society met Tuesday, May 27, at 7:30 p. m. at Minden Sanitarium at Minden, Louisiana. Dr. M. W. Hunter of Monroe, Louisiana, talked on The Diagnosis and Treatment of Coronary Occlusion.

NEWS ITEM

Dr. Roy Carl Young of Covington, La., addressed The National Association of Private Psychiatric Hospitals on The Role of the Private Psychiatric Hospitals in the Treatment of Alcoholic Addiction, at its Annual Meeting at the Pennsylvania Hotel, New York, on May 18, 1947.

Dr. Young together with several other of our local psychiatrists, attended the meeting of the American Psychiatric Association from May 19 to May 23, 1947.

Dr. George M. Haik, Head of the department of Ophthalmology at Louisiana State University presented a paper on Intra-Ocular Foreign Bodies before the American Medical Association at its recent meeting in Atlantic City, N. J.

Dr. Rufus Alldredge, Assistant Professor of Clinical Orthopedics at Tulane, was on the program of the American Orthopetic Association at Hot Springs, Virginia.

THE AMERICAN CONGRESS OF PHYSICAL MEDICINE

This organization will hold its twenty-fifth annual scientific and clinical session September 2-6 inclusive, at the Hotel Radisson, Minneapolis. Scientific and clinical sessions will be given the days of September 3, 4, 5 and 6. All sessions will be open to members of the medical profession in good standing with the American Medical Association. For information concerning the convention, address the American Congress of Physical Medicine, 30 North Michigan Avenue, Chicago 2, Illinois.

LOUISIANA - MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

The Louisiana-Mississippi Ophthalmological and Otolaryngological Society held its annual meeting at The Buena Vista Hotel, Biloxi, Mississippi, on May 5, 1947.

Dr. George Adkins of Jackson, Mississippi, President of the Society, presided over the meetings. The following program was presented:

The Rhinoplastic Operation and the Restoration of Nasal Function: Dr. Samuel Fomon, New York City.

Newer Trends in the Treatment of Ocular Diseases: Dr. Peter C. Kronfeld, Chicago.

The J. Raymond Hume Memorial Address: Meniere's Disease: Dr. Henry L. Williams, Rochester, Minn.

Allergic Problems Seen by the Ophthalmologist and Otolaryngologist: Dr. Ralph Bowen, Houston, Texas.

Dr. Noel Simmons of Alexandria, Louisiana was elected President, and Dr. Edley H. Jones, of Vicksburg, Mississippi was re-elected Secretary. The 1948 Convention will be held in New Orleans, Louisiana.

THIRD AMERICAN CONGRESS ON OBSTETRICS AND GYNECOLOGY

It has already been pointed out that a magnificent program is being prepared for this important meeting, of which Dr. Fred L. Adair of Chicago is general chairman and which will be held in St. Louis September 8-12. Not only is the medical profession to be represented but a recent release has come to the journal office saying that an extensive nursing program has been planned for this Third American Congress. Women in the nursing field from all over the country are scheduled to appear on the program.

NEW HOSPITAL IN METAIRIE

Dr. William Kohlman Gauthier, Director of the New Orleans-Metairie Hospital Foundation announced that a 30 bed general hospital will be opened in the latter part of July at 310 Codifer Boulevard, Metairie. Modern colonial architecture in design, the building incorporates the latest facilities and development in hospital equipment, including an inter-communication system that enables the nursing staff to be in constant reach of individual patients. The hospital is so constructed that expansion can readily be made.

The building is the only one in the New Orleans Area constructed specifically as a private hospital during the past 17 years, although the population during the same period has increased from 390,000 to 650,00 people.

The staff of the hospital will be organized according to the rules and regulations of the American Medical Society and the American Hospital Association. The organization will be that of an open staff hospital and all doctors who are members of local, parish or state medical societies are invited to utilize its facilities.

Certification as an accredited hospital by the American Hospital Association has been applied for. Also application has been made to handle indigent cases in certain circumstances.

INTERNATIONAL COLLEGE OF SURGEONS

A convocation and assembly of the International College of Surgeons, United States Chapter, will be held in Chicago September 28-30-October 1-4. The General Chairman of the meeting is Dr. Raymond W. McNealy. An excellent program will be provided for those who plan to attend this meeting.

MAJOR GENERAL BLISS NEW SURGEON GENERAL

It is with pleasure that we read of the announcement of Major General Bliss being appointed the new Surgeon General of the U. S. Army Medical Corps. General Bliss is a graduate of Tufts Medical College, he entered the service in 1911 and has since that time been continuously with the Medical Corps of the Army. General Bliss has had many important assignments. He has been Chief of Surgery at the Sternberg General Hospital, Manila, at Fort Sam Houston and at William Beaumont General Hospital, El Paso, Texas. In 1943 he was made Chief of Operations; a few months later Assistant Surgeon General, in which capacity he served until June 1946, when he was made Deputy Surgeon General.

At the same time it was announced that Brigadier General George E. Armstrong assumes the duties of Deputy Surgeon General.

Former Surgeon General Kirk retired from active duty and it is understood will practice in New York City. To General Kirk should go very much more credit than apparently has been given to him for the able way in which he conducted the Surgeon General's Office and the various medical services during the period of active fighting. Every student of the Army Medical Corps will appreciate that Kirk did a magnificent job. The tremendous reduction in morbidity and mortality figures show that Kirk was willing to assume responsibilities that would permit of the medical profession in this country to go ahead and do an unsurpassably good job in taking care of the sick and wounded soldier.

DR. LUCIEN AMARON LeDOUX

A reprint of an editorial written about Dr. Lucien A. LeDoux and which appeared in the Southern Medical Journal a few months ago has come to the office of the journal. Dr. LeDoux, as the reprint points out, has been active and busy in the affairs of organized medicine. Furthermore, he has been particularly interested in the Southern Medical Association, of which he is now president-elect. We extend to Dr. LeDoux all best wishes for a successful tenure of office.

CARE OF THE HUNGRY

CARE is a non-profit, government-approved food package service to Austria, Belgium, Czechoslovakia, France, Finland, Greece, Hungary, Italy, Norway, the Netherlands, Poland, Rumania, the American, British and French zones of Germany plus all of Berlin. It is composed of twenty-seven major welfare agencies concerned with sending aid to Europe. It has been termed by General Eisenhower, a "personal expression of international goodwill."

The CARE food package contains more than twenty-one pounds of meat, fats, sugar, milk, flour, chocolate, coffee and other essential foods which are practically unobtainable in Europe today. Operating through agreements with the European governments, CARE guarantees delivery of its parcels ration-free, duty-free, tax-free.

Total cost of a CARE food package is $10. Orders are airmailed to the designated countries in Europe and delivery is made from local European warehouses. Individuals and organizations can send CARE food packages to friends and relatives in Europe. Orders may be sent to CARE headquartes, 50 Broad Street, New York 4, N. Y.

NATIONAL FOUNDATION FOR INFANTILE PARALYSIS, INC.

"Again during these summer months outbreaks of poliomyelitis are making their appearance in many sections of the country. Last year 25,191 cases occurred in the nation, 382 of them within this state. No one can forecast how many cases will occur this year or how badly the communities in this area will be affected. Medical science, unfortunately, cannot as yet prevent an epidemic or even one case.

"Physicians in this area, as well as elsewhere, are aware of the multitude of problems poliomyelitis presents. Treatment of the disease is apt to be prolonged and extremely costly, requiring the services of many specialists. Too often the patient's family looks to the physician for advice and guidance far beyond the immediate problem of medical care.

"In times such as these it is helpful to physicians to know that there are others prepared to share these troublesome burdens. In addition to making possible epidemic aid, education, and scientific research, the National Foundation for Infantile Paralysis is pledged to assist financially those patients who require such help. Through their generous contributions to the March of Dimes, the American people have made this possible. Hospital bills, salaries for physical therapists and nurses, purchase of special equipment, and the many other changes which may comprise the essentials of good medical care may be paid for by the Chapters of the National Foundation when necessary. Local Chapters of the National Foundation are scattered throughout the United States. There is one in or near your own community. Your local health department can furnish you with the address of the Chapter nearest you."

THE NUMBER OF BIRTHS DURING THE FIRST QUARTER OF 1947

There were approximately 973,000 births registered in the United States in the first three months of 1947. This is almost 50 per cent more than the number of births registered in the first three months of 1946, and it is 29 per cent more than the number recorded the first quarter of 1943, the record year until 1946.

The death rate in the country, according to the figures of the National Office of Vital Statistics was 388,000, comparable with the first quarter of 1946.

PHYSICIANS WANTED

The office of the Journal has been informed that a physician is needed for general practice in Livingston, Louisiana. For detailed information communicate with Mr. Sartwell in that locality.

A physician is wanted by the Elizabeth Sullivan Memorial Hospital in Bogalusa. For detailed information contact Dr. E. E. Lafferty at that institution.

A physician is needed in Vacherie, Louisiana. For further information contact Mr. J. Richard Haydel, Adjutant, The American Legion, Wallace Post No. 268, Vacherie, Louisiana.

HEALTH IN NEW ORLEANS

The Bureau of the Census, Department of Commerce, reported that for the week ending May 17 there occurred in the City of New Orleans 145 deaths. Figures are not available as to racial distribution of these deaths. It might be pointed out, however that there were 25 more people dying in the city than the previous week and that the number of infant deaths from both races was 20. The following week, which terminated May 24, showed a better death record than the previous week. In this week there were 134 deaths; the number of infant deaths was seven. There are no figures available for the racial distribution of these deaths but in the previous week the figures have become available and show that 96 of the deaths were in the white portion of the population and 49 in negroes. There was a very sharp reduction in the number of deaths amongst the citizenry of New Orleans for the week which closed May 31, only 93 deaths being reported, 61 of which were white, 32 negro and five deaths occurred in children under one year of age.

There was a very sharp increase in the death rate in the city for the week which closed June 7. The increase was almost 50 per cent above that of the previous week. Of the 154 people who expired in New Orleans 100 were white and 54 were non-white. Nine of the deaths occurred in infants.

INFECTIOUS DISEASES IN LOUISIANA

The weekly morbidity report for the State of Louisiana for the week which ended May 19, the following diseases were reported in numbers greater than 10: Tuberculosis for the first time in a long while led all deaths with 69 instances reported, followed by hookworm infestation with 59, cancer with 33, measles with 23, influenza 22, unclassified pneumonia 18, malaria 17, whooping cough 16, pneumococcic pneumonia 14, septic sore throat 12, other forms of tuberculosis and chickenpox 11 each. During this period of time there was one case of poliomyelitis reported which originated in this state and one case which was contracted outside the state. The following week, which closed May 17, found cancer leading the list of reportable diseases with 53 cases. This in turn was followed by 34 cases each of measles and pulmonary tuberculosis. There were 31 cases of unclassified pneumonia, 17 of chickenpox, 13 of whooping cough and 11 of pneumococcic pneumonia. There were no cases of poliomyelitis reported this week nor were there any unusual or rare diseases. For the week which came to a close on May 24, pulmonary tuberculosis again led all other reportable diseases with 62 cases listed at the Louisiana State Department of Health. Next in order of frequency was unclassified neumonia with 29 cases, cancer and whooping cough with 21 each and measles with 19. The only unusual disease, at least for these days and times, was a case of smallpox discovered in Sabine Parish. The last week in the month was a remarkably healthy week; there were only three diseases reported in numbers greater than 10, namely measles with 27, pulmonary tuberculosis with 19 and cancer with 18.

MONTHLY MORBIDITY FOR VENEREAL DISEASES STATE OF LOUISIANA

Month Ending April 30, 1947

	Total This Month	Total Previous Months	Total To Date 1947
Chancroid	43	178	221
Gonorrhea	1269	3800	5069
Granuloma inguinale	19	38	57
Lymphopathia venereum	7	26	33
Syphilis	949	3040	3989

BOND-A-MONTH

The following article is published at the request of the Treasury Department, U. S. Savings Bonds Division.

"CALL ON SOME DOCTORS, SON . . ."

The apple-cheeked investment salesman, freshly weaned from college, sets forth in the world with two pieces of equipment—a brief case for that dignified look, and some sound advice from his elders.

"The old-timers in stocks and bonds slap him on the back and give him the advice they had when they were young:

"Go call on some doctors, son."

It *is* sound advice. Doctors, too often, are good prospects for bluesky investments. When it comes to slick trading in securities, they haven't the time to investigate. They're scientists, analysts of human ailments, artists of the operating room, travellers in the night, worriers, dreamers, thinkers and curers. But not financiers.

Diagnose an illness? Sure. But when a thousand dollars could be made or lost on a one-point change in Giltedge Preferred, they're busy; a child's appendix has ruptured.

Buy the best X-ray machine at the right price? Probably. But skillfully manipulate industrial investments to put their children through college? Or to take care of the family when the old ears are not so sharply attuned to the stethoscope, hands not so sure on the scalpel? Not very often.

A doctor is a busy man and a hopeful one—hopeful that the fees will take care of the future. True, some doctors are wealthy, or near it. To most, wealth is just something someone else enjoys. But many doctors, lulled by the crowded waiting rooms which went hand-in-hand with the war-born doctor shortage, figure they'll be pretty well set for retirement, the way fees are piling up.

Yes, the doctor needs a "bullet-proof" investment, and the United States has provided it in Savings Bonds. We all know that, but just to make it available isn't enough.

Most doctors need something more. In business matters they need a string tied around a finger. The Government is offering this to them in the new Bond-a-Month Plan. Your bank ties the string, gives it a yank every month. And all the doctor has to do is leave it there. Wilson Mizner said: "The gent who wakes up and finds himself a success hasn't been asleep." As to planning his future, once the doctor has invested in Bond-a-Month, he *can* give all his attention to his important work.

Before getting down to what the Bond-a-Month Plan can do for the non-salaried doctor, let's take a look at some of the facts in the case of the average practitioner. He's busy, more of late than ever before. His routine is hospital calls, perhaps an operation or two, office hours, house calls, office records, telephoning, more calls after dinner. Every so often a baby fails to realize the doctor has a schedule, or someone breaks a leg, or gets measles. Even the specialists work no 9-to-5 day.

The U. S. Department of Commerce has made studies of doctors' incomes, based on reports of a sample of the 129,000 men and women in private practice in 1940. The studies show that the income rises slowly to a maximum in the early 50's and then starts dropping. From 35 to 54 is the real money-making period.

At 35, most doctors have begun to pay off their starting-in-business debts, have built up a small neighborhood practice and are becoming known. Their practice grows with ability. By the time they are 54, other doctors, young and vigorous, have come in with new methods, machines, theories. They make inroads into the established practice of the veteran. The older man no longer so willingly drives out into the country on sick calls. Office hours are shaved a little at the start and the end of the day. There are fewer operations.

And somehow, without the doctor's really knowing why, the bank balance doesn't hold up the way it used to.

To come as close as we can to keeping the horse in front of the cart, let's see what Bond-a-Month will do and then explain why this is the solution to the doctor's problem of saving for future security.

Bond-a-Month opens systematic saving through Government bonds to anyone with income and a checking account in a bank. Until now this was available only through Payroll Savings. It operates this way:

The depositor who wishes to buy a bond each month signs a card authorizing the bank to deduct the purchase price from his checking account. The bank issues the bonds and delivers them to the customer monthly. The periodic bank statement shows payment for the bonds.

And from the first and the only time the doctor signs his authorization card, he has nothing else to do except open the envelopes the bank sends him with the bonds inside.

What does the doctor need?

1. He needs some sort of arrangement for his financial future because, according to studies of his profession, incomes of physicians are much more responsive to change in the national income than are the incomes in other professions. If the national income drops and patients no longer can afford to call on the doctor so often or to pay him as quickly, a doctor's bankbook will feel the change.

2. In most cases, the doctor has no social security or pension to fall back on. Thus, he needs something to serve as an old-age reserve.

3. He needs simplicity—an arrangement which does not call for continual checking, manipulating, buying and selling.

4. He needs safety. He cannot afford to take the risks which must be protected by constant market vigilance, by buying and selling strategically.

A savings bond plan should be the foundation upon which the doctor builds his security. There is no safer investment in the world than Savings

Bonds. There is no riskless investment which pays such a guaranteed return.

Consider:

If you invest monthly under the Bond-a-Month Plan	In five years You will have	In ten years You will have
$ 37.50	$ 2,319.00	$ 4,998.00
75.00	4,638.00	9,996.00
100.00	9,276.00	19,992.00
300.00	18,552.00	39,984.00

Here, for the doctor himself, are vitamins E, F and G, thoroughly tested and always compounded with interest. These "vitamins" ease common symptoms of post-middle age such as chronic worry and doubt. They are available at a bank near you. And with millions of current users, we can make this unusual guarantee: one and a third times your money back if you *are* satisfied.

THEOBALD ROBERT RUDOLPH
1877 - 1947

Dr. T. R. Rudolph died June 11, 1947, at the age of 70. Dr. Rudolph was a graduate of Tulane in 1902. For many years he was connected with the State Department of Health. He was a very well liked physician and will be missed by his many friends.

WILLIAM WORTHING CALHOUN
1868 - 1947

Dr. W. W. Calhoun died on June 11, 1947, at the age of 79. Dr. Calhoun practiced medicine in New Orleans for many years. He was a member of the parish and state medical societies until the time of his death.

EPHRAIM D. FRIEDRICHS
1877 - 1947

Dr. E. D. Friedrichs, who died on June 10, 1947, had been in ill health for the past several years and was retired from the active practice of medicine. Dr. Friedrichs graduated from the Tulane University School of Medicine and was at one time head surgeon of the old New Orleans Sanitarium which later became the Presbyterian Hospital.

JOHN ADEN LEWIS, SR., M. D.
1885-1947

Dr. J. A. Lewis, Sr., died on June 17, 1947 at the age of 62, after a protracted illness. Dr. Lewis was for many years on the staff of the Department of Medicine of Tulane University School of Medicine, and on the staffs of Baptist and Charity Hospitals. He practiced in New Orleans for 36 years and will be missed by his many patients. Dr. Lewis was a member of the Knights Templar, the Shriners, the Parish and State Medical Societies.

WOMAN'S AUXILIARY OFFICERS 1947-48

President, Mrs. James W. Warren, New Orleans.

President-Elect, Mrs. O. B. Owens, 1026 Bolton Ave., Alexandria.

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A GREETING FROM THE PRESIDENT

Through the medium of the Auxiliary page in the Journal I desire to extend a greeting to the members of the Woman's Auxiliary to the Louisiana State Medical Society. First, let me express my deep appreciation for the honor you have given me in making me your president for the next year. I accept the office in all humility, for I realize the responsibilities it carries with it and the inadequacy of my own capabilities. However, I think that willingness and determination to work can make up for many shortcomings, and these attributes I modestly claim. These, coupled with your cooperation which I earnestly solicit, can carry us a long way on the road to success.

To my immediate predecessor, Mrs. Arthur D. Long, I wish to express my deep gratitude for the assistance she has given me in preparing for the office and for the excellent condition in which she left the affairs of the organization. If our accomplishments even remotely approach hers, we shall feel that we have done well.

The members of the board chosen to work with us are all women of high caliber and I know I can depend upon their support. We pledge you to do our best to follow the fine example set us by former presidents and their boards.

MRS. JAMES W. WARREN,
President.

BOOK REVIEWS

The Lung: By William Snow Miller, M. D., Sc. D. 2d ed. 1947. Pp. 222. Springfield, Illinois, Charles C. Thomas. Price $7.50.

The second edition of this invaluable monograph on the anatomy of the lung, presents few changes from the first edition of 1937. However, this work was so complete and so valuable for reference, that few changes were necessary in this monument to the life work of William Snow Miller. A few illustrations have been improved and additional illustrations have been inserted in the chapters on the air spaces, lymphatics and the pleura. Some new studies have been added regarding the nerves of the visceral pleura. This book remains the classic study in English which should be in the library of everyone interested in the subject.

JULIUS LANE WILSON, M. D.

An Integrated Practice of Medicine: *A Complete General Practice of Medicine from Differential Diagnosis by Presenting Symptoms to Specific Management of the Patient*: By Harold Thomas Hyman, M. D. Philadelphia, W. B. Saunders Co. 1946 1184 illus, 305 in color, 319 differential diagnostic tables. 5v. Price $50.00.

The one stop shop, familiar to all Americans has finally come home to medicos. We will no longer need to search out many sources to obtain the medical "vittels" so essential to our medical ingestion, digestion and absorption. And what is even more important, it is all there in the form of extracts, concentrates and little-finger sketches, all obtainable with an economy of effort and time. Each generation has its system of medicine and ours is no exception, but with this revolutionary difference. It is "integrated". In the world of books, this one stands unique. The power of the atomic bomb to destroy may be compared with older instruments of destruction in magnitude. The striking superiority of the antibiotics and sulfonamides over the older bacteriostatic and -cidal agents lends itself to comparison. But of Hyman's work we read in a letter from the publisher and I quote, His "work cannot be compared with any other existing work because nothing like it has ever been published." The many important symptoms and complaints are presented in tabular form with a list of the diseases in which they are found. The several diseases with distinguished features, clinical and laboratory, of each, are numerated and by an adjoining page reference one may frisk through the four volumes with their 4131 pages, diagnostically and therapeutically refreshed. The mastery of the opus operandi may prove time-consuming, but the editor assures us that eventually you will be able to handle this system so skillfully and systematically that in no time at all you will know all that ordinarily takes so long to learn. You will no longer need to shop in many books and seek out the secrets of the specialists, for here, in close harmony and fully integrated you will find the most diverse subjects skillfully and symphonically arranged by the conductor-editor, Dr. Harold T. Hyman and his associates. To mention a few subjects is to call attention to the fact that this work has been conceived on a grand scale. Whether its execution has fulfilled the vision of the editor is something that remains to be proven. Only the reception by the profession will show whether this is to be a better mouse-trap or another case in which the mountain brought forth a mouse. Here you will read of physical diagnosis, physical medicine, psychosomatic medicine, prognosis, physician-patient relationship, endocrinology, minor surgery, gynecology, obstetrics, eye, ear, nose and throat, pharmacology, neuropsychiatry, office organization, the technics of specialist employment, pharmacology, dematology with many good Kodachrome illustrations. Illustrations are profuse but all have been copied from other sources. In keeping with the desire to make the work all-embracing the author has prefaced the diseases of the various body systems with a brief description of the anatomy and physiology of the organ-systems.

The reviewer feels that the sample readings were for the most part written simply, clearly and concisely. Most of the theories concerning unsolved problems have been omitted and the authors have been refreshingly frank and honest in debunking some of the solemn and opinionated utterances of the medical authorities. Yet, one cannot escape the conclusion that much of this terse and facile writing is the crystallization of years devoted to the theory and practice of medicine, surgery and their allied specialties and it is the reviewer's belief that such bald treatment of many important subjects will go for naught in enlightening the practitioner, especially in instrumental technics and electrocardiography.

The most serious criticism of the books is that they are bound volumes and this one fact would seem to lead to the disintegration of the integrated system. At one point, the author states in discussing certain therapeutic measures, that they are the result of his personal experience and he requests his reading public to write to him concerning their disagreement. What he expects to accomplish with such data is certainly not obvious as far as this system is concerned. It is to be hoped that the author does not contemplate an annual supplementary volume.

The index volume is an integral part of the

whole and one that must prove invaluable in the proper use of the system. In this day, with the tremendous advances in the science of medicine, one marvels over the necessity for the general practitioner who may be expected to be familiar with medicine, surgery and the specialties and to be fairly familiar with all procedures, diagnostic and therapeutic. He is a most courageous individual and most deserving of our praise and admiration. Beset by many tasks and with little leisure, such a system as this, encyclopedic in content, despite its shortcomings, may yet prove a worthy addition to his armamentarium.

I. L. ROBBINS, M. D.

Treatment of Arthritis and Rheumatism in General Practice: By Bernard Aschner, M. D. New York City, Froben Press, 1946. Pp. 340. Price $5.00.

This book is a review of the history of rheumatism and arthritis and obviously should be of great interest to the physician who is especially interested in historical medicine. The author takes the opportunity to criticize the present-day conception of arthritis, especially in regard to therapy. In Chapter III he gives, at great length, description of some of the older remedies which have been discarded by the modern physician in dealing with arthritis. It is his opinion that most of these old therapeutic measures are fare superior to those presently in vogue.

In Chapter IV there is a very interesting compilation of various arthritic cases which the author has had the occasion to treat during his professional career. Unfortunately this compilation is not arranged in any chronologic order or in reference to the etiology or treatment of the disease. These cases are interesting only from the standpoint of therapy which he has relied upon without the use of modern therapeutic agents.

One would presume from the expressions contained in the book that the treatment of arthritis has not been progressive and that we should revert to the use of drugs and the conception of etiology based on early historical clinical facts and data.

P. T. TALBOT, M. D.

A Primer for Diabetic Patients: By Russel M. Wilder, M. D., Ph.D., F. A. C. P. 8th ed. reset. Philadelphia, W. B. Saunders Company, 1946. Pp. 192. Price $1.75.

This very valuable little book should be a big help to all diabetics who are willing to study it. That Dr. Wilder expects his patients to adhere to a prescribed regimen studiously is indicated by the questions at the end of each chapter to be answered by the patient.

The conventional arrangement is followed. Various chapters describe the clinical features of the disease, examinations of the urine for sugar and for diacetic acid, insulins and their administration, the complications of diabetes, and the method of planning a diet and actually preparing menus for daily use. These chapters are sound and offer a wealth of practical advice.

One objection may be raised. In discussing mixtures of unmodified and protamine zinc insulin, the author gives detailed instructions for varying the proportions of these two insulins on the basis of the daily fractional urinalyses. It is doubtful that the average patient should be encouraged to make such changes which require the skill of the physician.

The book is clearly printed and well illustrated by a number of photographs and sketches. It is highly recommended.

SYDNEY JACOBS, M. D.

PUBLICATIONS RECEIVED

Froben Press, New York: Care of the Breast by Else K. La Roe, M. D.

Grune & Stratton, New York: Gastritis by Rudolph Schindler, M. D., F. A. C. P.; X-Ray Diffraction Studies in Biology and Medicine by Mona Spiegel-Adolph, M. D., and George C. Henny, M. S., M. D.

Medical Authors' Publishing Co., Chicago: Proceedings of the Eleventh Annual Convention of the National Gastroenterological Association, edited by Samuel Weiss, M. D., F. A. C. P., D. Sc.

W. B. Saunders Company, Philadelphia: Diseases of the Chest Emphasizing X-Ray Diagnosis by Eli H. Rubin, M. D., F. A. C. P., F. C. C. P.; Diseases of Metabolism by Garfield G. Duncan, M. D.; Gynecology Including Female Urology by Lawrence R. Wharton, Ph.B., M. D.; A History of the American Medical Association 1847 to 1947 by Morris Fishbein, M. D.; Surgical Pathology by William Boyd, M. D., Dipl. Psychiat., M. R. C. P. Edin., F. R. C. P. Lond., L. D. Sask., M. D. Oslo, F. R. S. C. Professor of Pathology, The University of Toronto; A Textbook on Medicine by Russell L. Cecil, A. B., M. D., Sc.D.

University of Chicago Press, Chicago: Curare by A. R. McIntyre, Ph.D., M. D.

Year Book Publishers, Inc., Chicago: Office Immunology edited by Marion B. Sulzberger and Rudolf L. Baer.